Recent Literature on the
Non-Auditory Effects of Noise:
The Primary Emphasis on the
Cardiovascular System

October 26, 1977

Prepared for

U.S. Environmental Protection Agency
Office of Noise Abatement and Control

by

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Selected Articles Processed on Other Non-Auditory Effects of Noise
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* English translation and original article included
Introduction

This compilation is the result of a literature search for recent articles (primarily 1975-1977) on the non-auditory physiological effects of noise. The main area of interest in this project was the effects of noise on the cardiovascular system. A list of the investigators in the field of noise effects who provided additional information for this project has been included. The articles in the bibliography that were acquired and analyzed will be used in future work by the Environmental Protection Agency on the non-auditory effects of noise.

Informative abstracts have been completed for the 21 most relevant studies concerning the cardiovascular effects of noise. In addition to the abstract, a form summarizing the experimental design and results, including evaluations and comments, has been provided for each abstract. The form was designed to provide an easy means for comparing and analyzing the studies. The abstracts, forms, and accompanying articles are arranged alphabetically by author in sections 1-21 of this volume.

A summary form only was completed for each of 13 additional articles that cover areas other than the cardiovascular effects of noise. These studies deal with other significant non-auditory effects, especially on the endocrine system, including catecholamine excretion and plasma cortisol levels. The forms and copies of the original articles are included in sections 22-34. Both time and money limitations prevented such processing of the other 78 articles in the bibliography. Copies of the majority of these articles have been acquired by the Environmental Protection Agency.

Some of the copies of the original articles accompanying the abstracts and summary forms are of poor quality, although they are legible. They were the best copies available at the time of printing.
Bibliography
(see page 9 for footnotes)


† Hanson, J. D. et al. (Dep. Psychol., Univ. Wis., Madison, Wis. 53706, USA). The effects of control over high intensity noise on plasma cortisol levels in Rhesus monkeys. Behav Biol 16(3):333-340, 1976.


Kharkovenko, N. M. Physiological and hygienic evaluation of the working conditions of operators in the oxygen converter smelting of steel. Gig Tr Prof Zabol (11):46-48, 1973. (in Russian)


Krasin, J. The adverse effects of noise on the flying personnel of the civilian aviation of the GDR with special reference to age and time of exposure. Z Gesamte Hgy 22(5):312-318, 1975. (in German)


** Marinyako, A. Z. and V. V. Lipovoy. An estimate of the total time of individual noise effects in hygienic evaluation of intermittent noises. Gig Tr i Prof Zabol 2:15-18, 1975. (English translation)


** Peterson, E. A. et al. (Division of Auditory Research, Dept. of Otolaryngology, University of Miami, School of Medicine, Miami, Florida). Continuing studies of noise and cardiovascular function. 1976. (unpublished paper).


Tsapko, V. G. Combined effect of organophosphorus pesticides and noise on the bodies of warm-blooded animals. Gig. i Sanitaria 5:32-34, 1976. (in Russian)

† Vander, A. J. et al. (Dept. of Physiology; University of Michigan). Effects of noise on plasma renin activity in rats. (unpublished paper).


Footnotes

* copy of article not available at Informatics Inc.

** abstract, summary form, and original article in sections 1-21.

† summary form and original article in sections 22-34.
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**SUMMARY FORM FOR STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

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**Institution and address where research was performed:**
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**Citation:**

<table>
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**Type & duration of experiment:**
Type: Retrospective case study
Duration: Results of annual physicals for past 7-8 years were examined.

**Purpose for study:**
To test for measurable non-auditory physiological changes in workers exposed to high occupational noise levels.

**Description of test groups (subjects.):**
Two groups: 1) Tests - 22 professional pilots in the FAA (male, 40-60 years old).
2) Controls - 29 non-flying FAA executives (male) of same age and socioeconomic status as the tests.

**Control of other stresses:**
None - field exposure to noise while flying.

**Noise Stimulus:**
Source: Aircraft noise (twin-engine transport or business aircraft; 4-engine transport; jet transport).
Spectral characteristics: Spectra included for cockpit noise in four types of planes.
Noise level: Noise levels (shown graphically) often exceeded Damage Risk Criteria.
Length of exposure: 6000 hours or more flying time.
For trials: Not applicable.

**Statistical Methods:**
A Randomized Complete Block Design used; t-test compared results for the two groups; F-test compared results within each group.

**CVS Response Measured:**
Heart rate using EKG records; blood pressure - mean systolic and mean diastolic levels.

**Nonauditory effects:**
CVS: No changes due to noise; fluctuations in diastolic blood pressure and heart rate in the pilots; increased systolic blood pressure in all subjects (probably due to age); decrease in heart rate in the controls.
Glucose: Both serum cholesterol levels and mean serum glucose levels declined with each measurement - no changes due to noise.

**Author's conclusions:** Results of audiograms showed noise-induced hearing loss in the professional pilots, but not in the control group. A comparison of the health records of the test and control groups did not show that occupational noise exposure produces long-term non-auditory physiological effects. The negative results could be due to insufficient noise levels or to lack of highly sensitive measurements of physiological responses.

**Evaluation & Comments:** The lack of significant non-auditory effects may be due to the intermittent exposure of the pilots to high noise levels, in contrast to that of industrial exposure, such as auto workers. Not only is the daily exposure of factory workers more continuous, but also the number of hours of exposure would be much greater than for pilots.

A retrospective study of the non-auditory physiological effects of noise was done in a test group of 22 professional male pilots, aged 40 to over 60 years. Heart rate, blood pressure, blood glucose, and serum cholesterol measurements from annual physicals (in an executive physical program) during the past 7 to 8 years in the pilots were compared to those of a control group of 29 non-flying male executives of similar socioeconomic level and age range. Both the test and control subjects were employed by the FAA (Federal Aviation Administration). The yearly means for each of the four physiological parameters measured were analyzed for changes over time. Changes within each group and between the two groups were analyzed using the F-test and the t-test respectively. The pilots, each with over 6000 hours flying time, were often exposed to noise levels exceeding the Damage Risk Criteria (DRC) of CHABA (the National Academy of Sciences Committee on Hearing Bioacoustics and Biomechanics). Spectra of the cockpit noise for four types of aircraft (twin-engine business and transport, four-engine transport, and jet transport) are included. Audiometric histories showed noise-induced hearing damage in the pilots, but not in the controls. Although some changes occurred in the physiological parameters measured, none of the changes were the result of occupational noise exposure. There was an increase in systolic blood pressure in both tests and controls, which was probably due to age. Changes in the other parameters included insignificant diastolic blood pressure fluctuations, decreased heart rate in the controls, fluctuating heart rates in the tests, and declining glucose and cholesterol levels in both tests and controls. The authors note that the decline in cholesterol levels may have been due to changes in dietary and exercise habits of the FAA personnel due to their involvement in an executive physical program.
Certain Non-auditory Physiological Responses to Noises

J. E. BROWN III,* M. S. R. N. THOMPSON, Ph.D., and E. D. FOLK, Ph.D.

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The non-auditory physiological effects of extended exposure of pilots to high intensity noise were investigated. The health records of 22 professional pilots (FAA) were examined for recorded measurements of heart rate, systolic and diastolic blood pressure, serum cholesterol, and glucose. These data were compared to records of the same measurements from 29 non-flying FAA personnel of the same age, social and economic status as the pilots. The yearly means for each parameter were analyzed for changes with time, within the same population, and for differences between the two study groups. Auditory noise histories were tabulated and compared, and noise levels inside aircraft were determined. Although it was demonstrated that FAA pilots were exposed to high occupational noise levels there was no indication that their exposure produced any significant non-auditory physiological response.

Introduction

The short-term non-auditory physiological effects of noise on man are relatively easy to measure and are reasonably well documented in the literature. It is generally agreed that sudden non-repetitive noise can cause alterations in voluntary muscle activity,1 vasodilatation of peripheral blood vessels,2 changes in heart rate4 and blood pressure,5 changes in respiration,1 alterations in gastrointestinal tract activity,1,2 and certain endocrine6 and biochemical changes.7,8 Not always agreed upon are the magnitude, direction and persistence of these startle responses. Nor is there any real accord in regard to the significance of these responses as precursors of non-auditory effects upon individuals exposed to noise for long periods of time.9 A review of the literature reveals that much more clinical and epidemiological evidence must be gathered before any valid conclusions can be made.

The purposes of this research were: (a) to select a population of workers known or suspected to be exposed to high occupational noise levels; (b) to confirm that they did experience such exposure; and (c) to investigate the possibility that their exposure may have produced some measurable non-auditory alterations in physiological function. It was anticipated that this retrospective study, though subject to the usual problems of "alter the fact" data gathering and interpretation, would provide some valuable baseline information for the design and conduct of subsequent prospective studies.

Procedure

Selection of a test population was not a difficult decision for some rather obvious reasons. First, the Federal Aviation Administration (FAA) has a fleet of approximately 100 aircraft of many types used for a variety of missions such as monitoring of airways navigation aids, inspection of instrument landing systems, flight training, and logistics. Second, many of the pilots who operate these aircraft are based at the FAA Aeronautical Center in Oklahoma City. Next, most of these pilots have logged many thousands of hours in all types of aircraft. And finally, all of them are given annual physical examinations for FAA medical certification in the
An "executive" based physical examination program conducted at the Center provided control subjects. Most of the pilots are in this program and receive their medical certifications with their executive physicals. The two groups were essentially of the same socio-economic status.

Each physical examination included a complete history, routine laboratory and radiographic procedures, electrocardiography (ECG), audiometry, and biochemical tests for alkaline phosphatase, cholesterol, glucose, protein bound iodine, thymol turbidity, triglycerides and uric acid.

The data assembled and analyzed in this study were selected from the individual health records of the participants in the program. All physical examinations were conducted by the same flight surgeon and all clinical laboratory procedures were accomplished by the same team of chemists and technicians throughout the study period.

In order to accumulate as many observations as possible, only those individuals with at least eight years' participation in the program were selected. Twenty-nine pilots meeting this criterion were located and it was subsequently determined that each had accumulated 6,000 hours or more of flying time. The 29 control subjects were non-flying executives with at least eight years' participation in the program.

The health records of each person were examined for recorded measurements of heart rate, systolic and diastolic blood pressure, cholesterol and glucose. These data were analyzed for changes with time and comparisons were made between the test and control populations.

A search of the records revealed that not all subjects received a physical each year. Therefore, means of the eight most recent recordings of heart rate and blood pressure and the four most recent measurements of cholesterol and glucose were used to test for changes with time. There was one exception: only seven mean values of heart rate were available in the control group, and consequently, comparisons between the test and control populations were made on the basis of seven consecutive heart rate determinations. A Randomized Complete Block Design with subjects as blocks was used for the statistical analyses.

Discussion

Noise Exposure Data

The nature of the noise exposure of the test population has been studied extensively by the F.A.A. and noise levels in crew compartments of all agency aircraft were obtained from various unpublished agency projects. In Figures 4 through 6, noise spectra were plotted for comparison with the National Academy of Sciences Committee on Hearing, Bioacoustics and Biomechanics (CHABA) Damage Risk Criteria (DRC) contours. These spectra were obtained for octave band frequency analyses made on the flight decks of the various aircraft at cruise conditions. During takeoffs, pilots are exposed to higher noise levels, much higher in piston-driven aircraft.

The 8-hour DRC contour was equalled or exceeded by noise levels in the cockpits of all of the aircraft except the jet transports. In five aircraft the 8-hour DRC contour was equalled or exceeded, and in one air-

![Figure 1: Coopert noise in twin-engine business aircraft.](image-url)
The 2-hour DRC contour was violated. These charts offer evidence that the test population was exposed to high occupational noise levels and audiometric evaluations confirmed this fact.

**Figure 2.** Cockpit noise in twin-engine transport aircraft.

**Figure 3.** Cockpit noise in four-engine transport aircraft.

**Figure 4.** Cockpit noise in jet transport aircraft.

**Auditory Effects**

Since both temporary and permanent alterations of hearing in response to excessive noise occur most dramatically at 4,000 Hertz (Hz) and, to a lesser extent, 6,000 Hz, hearing levels in these frequencies were analyzed for evidence of such alterations in the pilot population. Also, while advanced cases of noise-induced hearing loss cannot be distinguished from presbycusis, in less severe cases a 4,000 Hz "notch" (marked increase in hearing level at 4,000 Hz) followed by good residual hearing in the higher frequencies is diagnostic of noise-induced hearing loss in those persons with a history of noise exposure. Presbycusis generally first affects the higher frequencies and gradually proceeds to the lower.

The study population were placed in age groups of 40 to 49 years, 50 to 59 years, and 60 years and above. The hearing levels in decibels, referenced to 1951 American Standards Association (ASA) audiometric zero, were recorded for the frequencies of 4,000 and 6,000 Hz as found on the first and last audiograms in the individual health records. The mean hearing levels were determined for each age group at each of the two frequencies, and these data were used to compare hearing loss in the pilot and control populations. The results of these comparisons are contained in Table 1.

In the 40 to 49 age group, the pilots were found to have somewhat more acute hearing in the initial audiogram at both frequencies, though in the last the hearing level was nearly equal to that of the control group. The pilot group demonstrated a greater relative increase in hearing level in both test frequencies than did the controls during the study period.

The age group 50 to 59 also demonstrated an apparent noise-induced hearing loss in the airmen. The pilots were found to have a substantially greater hearing loss in the first audiogram at 4,000 Hz while the loss at 6,000 Hz was nearly the same in both
TABLE I

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population groups. As in the 40 to 49 year age group, the pilots demonstrated a greater relative increase in hearing level than did the control group.

The pilots also experienced greater hearing loss than the controls in the age groups 60 and above. However, the relative increase was found to be much the same.

Audiometric data for the pilots in the age groups 50 to 59 and 60 and above demonstrated the characteristic noise-induced 4,000 Hz notch in the initial tests.

These results did not differ greatly from those of Kronower and Somerville. While their study did indicate a greater degree of hearing loss in all age groups for both test and control subjects, the amount of difference between test and control subjects was almost identical.

Non-Auditory Effects

For examination of non-auditory physiological effects of noise, test and control subjects were age-grouped as previously mentioned. However, the noise exposed group contained only three pilots aged 40 to 49 and only four pilots were 60 or older. Since these age groups were believed too small to yield reliable results, only those aged 50 to 59 were given detailed consideration. Consequently, the noise exposed population contained 22 pilots and the control included 29 individuals.

A. Heart rate

The heart rates, taken from ECG records, are presented in Figure 5. The mean values of heart rate were found to fluctuate considerably without establishing a trend either toward increase or decrease. A statistical test comparing the means (F-test) indicated that the magnitude of these fluctuations was statistically significant within each population (p < 0.05), and a test demonstrated a significant difference in the heart rates in the two populations (p < 0.01).

Also, the more pronounced total decrease in the heart rate in the controls and the tendency to fluctuate in the pilots were in contrast with the general decrease in the pulse in response to audogenic stress reported in the literature.14
B. Systolic blood pressure

The mean systolic blood pressure (Figure 6) in the test subjects rose slightly in the early determinations, but dropped in the fourth determination. This decrease was followed by an increase which continued through the final determination. The mean systolic pressure of the controls declined through the first four determinations but increased through the last four. The magnitude of change within both groups was found to be statistically significant (p < 0.01), while the t-test indicated no significant difference between the two. The observed increase may have been due to aging.

C. Diastolic blood pressure

The mean diastolic blood pressure (Figure 7) showed no appreciable change with time, but fluctuated in the same manner as the heart rates. Tests for changes within populations and between the two populations were not significant.

D. Cholesterol

The mean serum cholesterol level (Figure 8) declined slightly with each successive determination in both pilots and controls. The degree of this decline, when evaluated by the F-test, did not prove significant and no difference between the two populations was indicated by the t-test. This decrease in the level of cholesterol was the opposite of the effect noted in earlier studies and was due, perhaps, to an increased awareness, on the part of those involved in the executive physical program, of the effect of diet and exercise on the cardiovascular system.

E. Glucose

The mean glucose level in the blood (Figure 9) also tended to decline with each determination, though the final value in the
Summary and Conclusions

(1) Changes observed in the heart rate over a seven-year period, though statistically significant within population and between populations, did not show a decline in rate resulting from noise exposure nor were those changes of sufficient magnitude to be considered biologically important.

(2) Changes observed in the systolic blood pressure, though statistically significant in the magnitude of change within populations, did not reflect a significant difference between the two populations. These changes appeared to be a normal increase with age rather than an effect of noise.

(3) Changes observed in the diastolic blood pressure were not significant, either statistically or biologically.

(4) Cholesterol levels decreased with successive determinations in both populations. This decrease might have been due to changing dietary and exercise habits.

(5) Glucose levels decreased with successive determinations in all cases except the final determination in the controls. Although statistically significant alterations in glucose levels were found within each population, no significant difference between the populations was discovered. Therefore, noise did not produce a statistically detectable change in glucose levels in the pilots.

(6) The noise exposed population demonstrated characteristics of noise induced hearing loss that were not found in the control population.

(7) Noise levels inside certain types of aircraft often exceeded levels of damage risk.

(8) Three general conclusions concerning the results of this study include: (a) either noise does not produce long-term non-auditory physiological responses, or (b) the noise levels to which these pilots are exposed are not of sufficient intensity to produce such responses, or (c) the method of measuring the physiological parameters involved are not of sufficient sensitivity to detect such responses as might be precipitated by noise.

References


Accredited Laboratories

The Board has approved the following laboratories as having achieved accreditation under the AIHA program.

Eastman Kodak Company
Health & Safety Laboratory
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1669 Lake Avenue
Rochester, NY 14650
Robert L. Raleigh, M.D., Director

Employers Insurance of Wausau
Environmental Health Laboratory
2000 Westwood Dr.
Wausau, WI 54401
Robert E. Hawkinson, Director

Massachusetts Institute of Technology
Industrial Hygiene Laboratory
77 Massachusetts Avenue
Cambridge, MA 02139
Richard J. Chambertain, Director

Ohio State Division of Public Health Laboratories
Industry Chemistry Section
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<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tr>
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**Purpose for study:**
To record statistically significant changes in blood pressure, pulse rate, and pulse pressure in humans after exposure to industrial type noise.

**Description of test groups:**
1 group - each subject served as his own control
20 healthy adult human volunteers, age 17-49 years, 11 females, 9 males
Activity of subjects - resting, seated

**Control of other stressors:**
Laboratory conditions used

**Noise Stimulus:**
Source: random noise generator
Spectral characteristics: broad band (spectral noise included)
Length of exposure: 1 hour noise

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<td># of tests</td>
<td>1-2 hour control (quiet) run per subject on different days</td>
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**Statistical Methods:**
Test and control values compared using the standard paired t-test.

**CVS response measured:**
Blood pressure (BP), pulse rate, pulse pressure, EKG recorded at 2 min. intervals.

**Physiological effects:**
CVS - transient changes occurred (attributed to chair rest, not noise exposure)
Other - A temporary threshold shift (TTS) was produced by 91 dBA noise.

**Author's conclusions:**
No statistically significant changes in the CVS responses measured occurred in subjects exposed to 1 hour of 91 dBA noise. Two hours of chair rest did produce significant changes - decreased systolic BP and pulse pressure, decreased heart rate and increased diastolic BP under both quiet and 91 dBA noise levels.

**Evaluation & comments:**
The exposure time (1 hr.) was too brief to indicate the effects of industrial noise conditions on the CVS. The amount of the TTS due to noise was not reported.

The cardiovascular effects of one hour exposure to 91 dBA broadband noise were studied in 20 healthy adult volunteers, aged 17 to 49 years. The 11 women and 9 men each served as their own controls. A 2 hour test period and a 2 hour control period were run on each subject on different days in an audiometric test chamber. The test runs consisted of 30 minutes quiet conditions, about 38 dBA, 1 hour of 91 dBA noise from a random noise generator, and 30 minutes of quiet (38 dBA). The control runs consisted of 2 hours of quiet (about 38 dBA). The subjects were seated in a chair and resting while in the test chamber. The blood pressure, pulse rate, pulse pressure, and electrocardiograms of the subjects were recorded automatically at 2 minute intervals in both the test and control runs. Audiograms were performed on the subjects during the pre- and poststimulus 30 minute periods in the test runs and during the same time periods in the control runs. The 91 dBA noise level was strong enough to produce a temporary threshold shift (TTS) of unspecified intensity in the subjects. The data was analyzed, using the standard paired t-test of the raw values for the subjects under both test and control conditions for corresponding units of time. No statistically significant changes due to noise were found in the cardiovascular parameters measured. The following significant changes were recorded due to the 2 hours of chair rest: slower heart rate, decreased pulse pressure and systolic blood pressure, and increased diastolic blood pressure. Graphs of the spectra under both the noise and quiet conditions are included. The raw data is presented in tabular form.
Blood pressure, pulse rate and pulse pressure were determined at 2-minute intervals on 20 healthy subjects while they were exposed to 91-dBA continuous broadband noise for 1 hour. Control data were obtained by replication without the noise. The noise produced no statistically significant changes in any of the cardiovascular parameters under study.

The effects of broadband noise on the cardiovascular system in normal resting adults

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Introduction
In 1970, Antociaglia and Cohen1 published a review of the then available literature on the extra-auditory effects of noise on man. Their paper was especially interesting because it revealed that this aspect of noise exposure had received little attention and that much of what had been done was controversial, contradictory, and in some instances, only conjectural. They noted that, in general, American noise experts in contrast to their European counterparts, believed that environmental noise presented but little threat to nonauditory systems. The prevailing view in this country at the time of their review was that adaptation to excessive noise minimized the likelihood of persistent alterations in physiologic function. The intent of their paper was "... to create active interest in and concern about this subject in those specialists having medical, hygiene, and safety responsibilities in industry..." and they concluded by calling for more study into the possible extra-auditory pathological effects of noise.

A review of the literature for this paper has shown that uncertainty surrounding the subject still persists, particularly in regard to the effects of noise on the cardiovascular system. It seems that investigators have generally chosen the convenient epidemiologic approach of retrospectively studying the incidence of cardiovascular disease among industrial workers exposed to noise or the less convenient but "quick result" method of measuring the startle noise effects on the cardiovascular system and extrapolating them to possible permanent effects. In both instances the inferences are somewhat tenuous.

There have been few controlled laboratory investigations into the effects of prolonged noise on the blood pressure and pulse rate of humans. Ponomareiko2 studied the influence of several noises of various intensities and spectral characteristics on the cardiovascular
functions in normal adolescent boys. He reported a lowering of the pulse pressure, a decrease in systolic pressure, an increase in diastolic pressure, and a decrease in pulse rate after 1 hour of exposure to 75-dB "wideband" noise. Identical tests, using the same subjects not exposed to noise, were used for control.

Terent'ev et al. exposed subjects to 1 hour of approximately 101-dB noise and reported decreases in heart rate and systolic and diastolic blood pressures. However, neither the spectral characteristics of the noise nor the activity of the subjects was reported, and no mention was made of control or the method of data analysis.

Lehmann and Tamm in 1956 published what is perhaps the most widely quoted article about man's cardiovascular response to prolonged noise. Eighteen healthy test subjects were exposed to various broadband 90-phon noises for 1 hour while blood pressures, pulse rates, and ballistocardiograms were recorded. The authors reported all their results in tables which indicated only that a particular parameter increased or decreased following exposure to noise. They concluded that there was little change in pulse rate and that pulse pressure amplitude showed a tendency to narrow, an occurrence attributable primarily to an elevation of the diastolic values. These observations were not substantiated by any numerical data.

This study was designed to try to measure any statistically significant changes (at the α = .05 level) in blood pressure, pulse rate, and pulse pressure (the difference between systolic and diastolic blood pressure) in subjects after prolonged exposure to industrial-type noise.

Procedure
Twenty healthy adult subjects, 11 females and 9 males, ranging in age from 17 to 49, were exposed to 91-dBA (rel. 0.0002 dyn/cm²) broadband noise in an audiometric test chamber for 1 hour, during which time systolic and diastolic blood pressures, pulse rates, and electrocardiograms were automatically recorded at 2-minute intervals. Each subject served as his own control when, on another day, the same cardiovascular parameters were measured in the chamber under "quiet" conditions (approximately 38 dBA).

The 91-dBA sound level was selected because it was, at the time this study was conducted, a value just greater than that permitted in American industry for an 8-hour workday without ear protection. This level was sufficiently intense to produce temporary damage to the ears in most subjects as detected by a temporary threshold shift (TTS) in their poststimulus audiograms. According to some physiologic theory, a stimulus great enough to produce a TTS should contain enough energy to "spill over" into another system, such as the cardiovascular system, and thereby produce a measurable alteration.

During the first 30 minutes of each test run, a 38-dBA sound level was maintained to adapt the subject to his environment and to perform the prestimulus audiogram, whereupon the noise was introduced through a 6" x 9" loudspeaker and adjusted to 91 dBA ± 1 dB as measured 3 inches from the subject's left ear. The spectra for both quiet and noise conditions are shown in Figure 1 for comparison with the noise used by Ponomarenko. The noise was produced by a General Radio Company random noise generator, set to the United States of America Standards Institute (USASI) mode, driving a 100-watt Bogen power amplifier. The noise was monitored and recorded continuously during all runs with a Bruel and Kjaer (B&K) microphone amplifier, type 2603, and a B&K graphic level meter, type 2305.

At the end of 1 hour of noise, the source was silenced and the poststimulus audiogram performed. The subject remained in the quiet chamber until the 2-hour run was completed.

Systolic and diastolic blood pressures were determined automatically by using a Nera bio-systems electrophysiomyomanometer, model ESG-300, and were recorded on one channel of a Model 5 Grass polygraph.

Figure 1 - Spectral analysis of: 1) noise used in this study, 2) noise used by Ponomarenko, and 3) "quiet" conditions of this study.

September, 1975
A standard limb lead I electrocardiogram was obtained from the arms by using large silver electrodes, and it was continuously recorded on a second channel of the polygraph.

A third channel recorded the pulse, which was detected by a piezoelectric crystal located over the left radial artery at the wrist where the artery is superficial.

All audiometry was performed with a Grayson-Studler recording audiometer, model 1703.

The complete experimental system is shown schematically in Figure 2 and illustrated in Figures 3, 4, and 5.

Results
For each of the 20 subjects, two 2-hour runs were performed, and these provided a total of 80 hours of test and control monitoring. Recording the designated cardiovascular parameters at 2-minute intervals yielded an unwieldy 9,600 observations, which were reduced to 3,200 mean values that were time dependent as follows: For each test and control run, there were originally 60 numerical values for each parameter measured. These were converted to 20 "means of size three" so that in plotting, the first data point represented the mean of measurement made at 2, 4, and 6 minutes into
the test runs and their corresponding time periods in the control runs. The five periods are designated in Figures 6 and 7 and described as follows: (A) the first 6 minutes of noise; (B) the central 12 minutes of noise; (C) the last 6 minutes of noise; (D) the first 6 minutes after cessation of noise (i.e., the start of the quiet period after the 1-hour exposure to noise); and (E) the final 6 minutes of a 2-hour run.

The change of the mean value of a parameter during each of these time periods from the mean value of that parameter during the first 30 minutes was determined for the test runs. Similar data were generated for the control runs. These test and control "change values," which represented both magnitude and direction of change, were compared by using the standard paired t test.

As shown in Table I, only two of the 20 t tests were significant at the \( \alpha = 0.05 \) level, and both occurred in the systolic blood pressure test.

The most important tests were the comparisons of the changes in blood pressure, pulse rate, and pulse pressure during period C, for by then the subjects had experienced the longest exposure to the test noise. However, none of the parameters revealed a statistically significant change.

The original null hypothesis (i.e., \( H_0 \): noise causes no statistically significant change in any of the cardiovascular parameters under study) was not rejected because the t values for all parameters for period C were too small for statistical significance at the \( \alpha = 0.05 \) level. When the null hypothesis is not rejected, the

![Figure 6 - Mean systolic and corresponding mean diastolic blood pressures based on raw data for all subjects during all experimental runs (20 control, 20 test).](image)

![Figure 7 - Mean pulse rates based on raw data for all subjects during all experimental runs (20 control, 20 test).](image)

- a run, and data point two was the mean of measurements made at 8, 10, and 12 minutes. This conversion was continued to data point 20, which was the mean of measurements made at 11, 11, and 12 minutes, the last 6 minutes of a run.

The raw data parameters of blood pressure and pulse rate, averaged for each point in time for all subjects for both control and test conditions, are presented graphically in Figures 6 and 7. It is readily apparent that blood pressure, pulse rate, and pulse pressure did undergo changes during the course of the 2-hour run.

To determine if the observed differences between the test and control runs were not merely chance occurrences, the observed changes were evaluated by using data representing five arbitrarily chosen time periods in

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Results of t testing on raw data.</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIME PERIOD</td>
<td>PARAMETER</td>
</tr>
<tr>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>A</td>
<td>Systolic BP</td>
</tr>
<tr>
<td>B</td>
<td>Systolic BP</td>
</tr>
<tr>
<td>C</td>
<td>Systolic BP</td>
</tr>
<tr>
<td>D</td>
<td>Systolic BP</td>
</tr>
<tr>
<td>E</td>
<td>Systolic BP</td>
</tr>
<tr>
<td>A</td>
<td>Diastolic BP</td>
</tr>
<tr>
<td>B</td>
<td>Diastolic BP</td>
</tr>
<tr>
<td>C</td>
<td>Diastolic BP</td>
</tr>
<tr>
<td>D</td>
<td>Diastolic BP</td>
</tr>
<tr>
<td>E</td>
<td>Diastolic BP</td>
</tr>
<tr>
<td>A</td>
<td>Pulse Rate</td>
</tr>
<tr>
<td>B</td>
<td>Pulse Rate</td>
</tr>
<tr>
<td>C</td>
<td>Pulse Rate</td>
</tr>
<tr>
<td>D</td>
<td>Pulse Rate</td>
</tr>
<tr>
<td>E</td>
<td>Pulse Rate</td>
</tr>
<tr>
<td>A</td>
<td>Pulse Pressure</td>
</tr>
<tr>
<td>B</td>
<td>Pulse Pressure</td>
</tr>
<tr>
<td>C</td>
<td>Pulse Pressure</td>
</tr>
<tr>
<td>D</td>
<td>Pulse Pressure</td>
</tr>
<tr>
<td>E</td>
<td>Pulse Pressure</td>
</tr>
</tbody>
</table>

September 1975

POOR COPY
The magnitude of the β (Beta) power of the test becomes extremely important, since the β power measures the probability of finding a difference between the test and control conditions if a difference is present. This difference must be specified before the β power can be determined.

The alternate hypothesis (Hα) was that noise will produce a ± 6 mm or greater change in blood pressure or a ± 6 beat-per-minute change in pulse rate. In every case the β power was greater than 97.5%, which indicated a high probability of detecting the stated difference between test and control conditions.

Among the several factors that can influence the levels of blood pressure and pulse rate are the emotional "set" of a subject on a given day and the season of the year. Consequently, it was decided to convert the raw data for each subject into percentage changes from the mean values, which would help to eliminate these additional sources of error. The results of this conversion are shown in Figures 8 through 11. A complete examination of the methodology used and the tabulated data are contained in the work by Cartwright. It is not statistically permissible to use the same type of paired t tests on the data after such manipulation. However, the graphs do accurately illustrate the behavior trends of the various parameters. It should be noted that under the conditions of both quiet and noise, the following occurred:

a. The systolic blood pressure decreased for about 30 minutes and then became stable. It increased, however, when the second audiogram was performed.

b. The diastolic blood pressure decreased for approximately 30 minutes, then started to increase and continued to do so throughout the experiment. The diastolic blood pressure did not seem to be affected by the second audiogram.

c. The pulse rate decreased rapidly during
the first 30 minutes and then decreased more slowly for the next hour. The pulse rate dipped at the time of the second audiogram, perhaps in a reflex manner in response to the systolic blood pressure increase at this point.

d. The pulse pressure decreased throughout the entire 2 hours of the experiment, but, as would be expected, it did show a transient increase at the time of the second audiogram. (The systolic blood pressure increased at this point and the diastolic blood pressure was unchanged; hence, the pulse pressure increased.)

Eight paired t tests were performed on the percentage change data comparing each parameter's initial value to its value at the end of the experiment. All eight were significant at the p = .001 level, which indicated that all the factors under study were time dependent in a highly significant manner.

Conclusions
The cardiovascular parameters of blood pressure, pulse rate, and electrocardiogram were continuously monitored during both noise exposure and quiet control conditions. Because these cardiovascular parameters are constantly changing, an attempt was made to detect any difference in these parameters under noise stress as compared to changes under quiet control conditions. From the results of this study, the following conclusions were made:

a. The exposure of resting normal adult volunteer subjects to broad-band noise of 91 dBA for a period of 1 hour produces no statistically significant change in any of the parameters at the α = .05 level. The β power of these tests was always greater than 97.5% when the alternate hypothesis (that noise would produce changes of ±6 mm of mercury in the blood pressures or ±6 beats per minute in the pulse rate) was applied.

b. Two hours of chair rest in normal subjects produce the following cardiovascular changes, all of which are statistically significant at the p = .001 level:
(1) a relative bradycardia;
(2) a decrease in the systolic blood pressure;
(3) an increase in the diastolic blood pressure; and
(4) a marked decrease in the pulse pressure.

c. In future investigations of this type, the fact that the blood pressure, pulse rate, and pulse pressure in normal resting human subjects do not reach a stable baseline condition in less than 11/2 hours must be kept in mind.

References
SECTION 3
<table>
<thead>
<tr>
<th>Description of test groups (subjects)</th>
<th>53 female rabbits, 2.7-3.2 kg: control group—15 rabbits—ordinary diet, test group—38 given 500 mg cholesterol in 5.0 ml. sunflower oil orally daily for 4.5-5 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type &amp; duration of experiment</td>
<td>5 months; laboratory study</td>
</tr>
<tr>
<td>Purpose for study</td>
<td>to study the effect of noise on lipid metabolism and on the development of atherosclerosis</td>
</tr>
<tr>
<td>Control of other stresses</td>
<td>laboratory conditions—the animals were very frightened by the noise</td>
</tr>
<tr>
<td>Noise stimulus</td>
<td>source: not given</td>
</tr>
<tr>
<td>spectral characteristics</td>
<td>3kHz</td>
</tr>
<tr>
<td>noise level</td>
<td>94-96 db</td>
</tr>
<tr>
<td>length of exposure</td>
<td>4.5 hr/day with 2-30 min. rest periods for 14 or 28 days</td>
</tr>
<tr>
<td>$\Delta$ of trials</td>
<td>4 groups given noise stimulation</td>
</tr>
<tr>
<td>Statistical Methods</td>
<td>student t-test</td>
</tr>
<tr>
<td>CVS Response Measured</td>
<td>(1) patho logical exam. of aorta, heart (2) platelet adhesiveness</td>
</tr>
<tr>
<td>Nonauditory effects</td>
<td>Increased platelet adhesiveness &amp; increased atherosclerotic changes due to noise. Increased hypercoagulation due to noise. Increased nonesterified fatty acids due to noise, ur. loss due to noise. Blood coagulation factors increased. Behavior—aggressive; fear</td>
</tr>
<tr>
<td>Author's conclusions</td>
<td>Noise stimulation caused increased levels of nonesterified fatty acids and blood coagulation factors (hypercoagulation). Hypercoagulation was enhanced by cholesterol feeding. Noise alone induced microscopic arterial necrosis and other atherosclerotic changes to a lesser degree in rabbits exposed to noise for 28 days than in those exposed for 14 days.</td>
</tr>
<tr>
<td>Evaluation &amp; comments</td>
<td>Noise may enhance the development of atherosclerosis in rabbits fed excess cholesterol levels. Individual differences between rabbits were observed. Relatively small numbers of rabbits in each group were studied.</td>
</tr>
</tbody>
</table>

The effects of noise and cholesterol on the development of atherosclerosis were studied in female rabbits weighing 2.7 to 3.2 kg. A total of 53 rabbits were divided into groups and treated as follows:

<table>
<thead>
<tr>
<th>Test Series</th>
<th>Group</th>
<th># of Animals</th>
<th>Treatment and Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Test</td>
<td>7</td>
<td>No cholesterol; noise 14 days</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>8</td>
<td>No cholesterol; no noise</td>
</tr>
<tr>
<td>II</td>
<td>Test</td>
<td>12</td>
<td>4.5 months cholesterol; 1 month no cholesterol; 14 days noise</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>8</td>
<td>4.5 months cholesterol; 1 month no cholesterol; no noise</td>
</tr>
<tr>
<td>III</td>
<td>Test 1</td>
<td>6</td>
<td>4.5-5 months cholesterol (noise for 1st 14 days)</td>
</tr>
<tr>
<td></td>
<td>Test 2</td>
<td>6</td>
<td>4.5-5 months cholesterol; (noise for 1st 28 days)</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>6</td>
<td>4.5-5 months cholesterol; no noise</td>
</tr>
</tbody>
</table>

The noise dose was 94-96 dB at 3 KHz. Daily exposure was for 4.5 hours with two 30 minute quiet periods every 1.5 hours. The daily dose of cholesterol used was 500 mg in 5 ml. sunflower oil, given orally. Blood samples were examined for changes in platelet adhesiveness and various coagulation factors, beta lipoprotein levels, unesterified fatty acid levels, and cholesterol levels. The hearts and aortae of the rabbits were examined for atherosclerotic changes. Major changes due to noise stimulation included increased platelet adhesiveness, higher levels of nonesterified fatty acids, and increased hypercoagulation. Noise both induced some microscopic atherosclerotic changes by itself and enhanced gross atherosclerotic changes in the coronary arteries caused by the atherosclerotic diets.
EFFECT OF ACOUSTIC STIMULATION ON LIPID METABOLISM,
INDICES OF THE BLOOD COAGULATION SYSTEM AND DEVELOPMENT
OF EXPERIMENTAL ATHEROSCLEROSIS IN RABBITS

By G.P. Deryagina, T.S. Sinitsina, T.V. Veselova

Fiziol.Zh. SSSR 62(8): 1171-81; Aug. 76

Laboratory of clinical and experimental cardiology
(I.E. Gapelina, director) Institute of Physiology
named after I.P. Pavlova, AN SSSR;
Laboratory of heart-vessel pathology (T.A. Sinitsina, head)
Institute of experimental medicine AN SSSR, Leningrad

The effect of sound caused an increase in the level of
non-esterified fatty acids in healthy rabbits over the course
of 14 days, as well as an increase of blood coagulation. Mor-
phologically we detected hypertrophy of the heart venal  
arteries, as well as focal points of necrosis in the myocardium. In the
case of rabbits with experimental atherosclerosis, subjected to
the effect of sound for periods of 14 and 28 days, despite the
high level of lipids in the blood and hypercoagulating shifts,
conditions were created facilitating resorption of the lipids
on the platelets of the aorta and coronary arteries. Moreover,
morphologically we also found hypertrophy and edema of the
vessel walls, focal points of necrosis, focal point and diffuse
adipose infiltration of the heart muscle.

Many experimental studies have shown that excessive stress
to the nervous system of different types increases vascular
permeability, facilitates an increase in the lipid fractions of
the blood and a precipitation of the lipids in the artery walls,
and causes hypercoagulation shifts in the blood coagulation
system [3,8,10,16].

By means of frequent changes in the stereotypes of rabbits
and dogs, P.S. Komul succeeded in causing atherosclerotic
changes in the aorta and in the large heart arteries [13].

However, the influence mechanism of overstrass of the
nervous system on the development of the atherosclerotic process
has not been finally explained. It is also not clear if the
character of the stress effect is important in the development
of the atherosclerotic process.

The purpose of the present work is to study the question
concerning the influence of extended overstrassing of the nervous
system, caused by sound stimulation, on the development of
experimental atherosclerosis in rabbits.

* This should be coronary.
Method

The work was carried out on 53 female rabbits with a weight of 2.7-3.2 kg. Of the 53 rabbits, 38 received 500 mg cholesterol in 5.0 sunflower oil per os daily for a period of 4.5-5 months, and 15 of the rabbits were kept on their ordinary feed. Some of the rabbits (34 of the 53) were subjected to the acoustical effect (94-96 db, 3 kilohertz) for a period of 4.5 hours per day (with two 30 minute interruptions for rest every 1.5 hours of the effect). The period of the sound effect was 14 and 28 days.

3 series of studies were carried out (Table 1). 15 rabbits were used in the first series, which were kept on their ordinary feed. 7 of them were subjected to the acoustical effect for a period of 14 days (group B). 8 of the rabbits served as a control group (group A). 20 rabbits were used in the second series, which, in addition to the usual ration, received cholesterol in the indicated dose for a period of 4.5 months. Then they were transferred over to ordinary feed ("for rest"). One month after removal of the cholesterol, 12 of the 20 rabbits were subjected to the acoustical effect for a period of 14 days (group B). 8 of the rabbits (group A) were not subjected to the acoustical effect. 18 rabbits were used in the 3rd series, receiving cholesterol for a period of 4.5-5 months. For 4 months from the start of feeding with cholesterol, 12 of them were subjected to acoustical effect against a background of continued giving of cholesterol (6 in a period of 14 days--group B, 6 for a period of 28 days--group B), and 6 (group A) served as a control group (these were not stimulated).

The blood was studied twice: immediately before the start of the acoustical effect and after it was stopped in conformity with the period of stimulation. Then the rabbits were sacrificed. The rabbits not subjected to the acoustical effect (group A) were examined in the same period of time.

We determined the indicators of lipid metabolism and the system of blood coagulation: cholesterol [22], total fraction of 3-lipoproteides [5], the non-esterified fatty acids [17], time of blood coagulation [20], recalcification time [21], tolerance of the plasma to heparin [23], prothrombin complex [14], fibrinogen [19], XIII factor [1], thrombin and heparin time [12], adhesiveness of the thrombocytes [18], fibrinolytic activity of the blood [2]. The blood stabilizer is 1.34% sodium oxalate. Its ratio in the blood is 1:4. The obtained numerical data were statistically processed by a different method with the use of the Student criterion t.

* This should be 5.0 ml.
Table 1.—Distribution of rabbits according to groups as a function of diet and sound stimulation.

<table>
<thead>
<tr>
<th>Test series</th>
<th>Cholesterol Group</th>
<th>No. of general</th>
<th>Period of diet feeding</th>
<th>Period of sound stimulation</th>
<th>Stimul.</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>A</td>
<td>8</td>
<td>--</td>
<td>--</td>
<td>14 days</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>7</td>
<td>--</td>
<td>--</td>
<td>14 days</td>
</tr>
<tr>
<td>II</td>
<td>A</td>
<td>8</td>
<td>4.5</td>
<td>--</td>
<td>14 days</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>12</td>
<td>Same</td>
<td>14 days</td>
<td>months</td>
</tr>
<tr>
<td>III</td>
<td>A</td>
<td>6</td>
<td>&quot;</td>
<td>--</td>
<td>14 days</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>6</td>
<td>&quot;</td>
<td>After 4 months</td>
<td>months</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>6</td>
<td>5 months</td>
<td>months</td>
<td>28 days</td>
</tr>
</tbody>
</table>

Table 2.—Change of the indicators (M±m) of lipid metabolism in rabbits without the effect (I) and under the influence of sound stimulation (II). (1) Animals; (2) Series; (3) Group; (4) Period of sound effect; (5) Cholesterol (in mg%); (6) Total fraction of 8-lipoproteins (in mg%); (7) Nonesterified fatty acids (in ml equiv/l); (8) Studies; (9) Without the effect; (10) 14 days; (11) 28 days.

<table>
<thead>
<tr>
<th>Group</th>
<th>Period</th>
<th>Cholesterol (M±m)</th>
<th>Total fraction of 8-lipoproteins (M±m)</th>
<th>Nonesterified fatty acids (M±m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>11 days</td>
<td>75.4±11.9</td>
<td>72.5±14.4</td>
<td>70.0±13.7</td>
</tr>
<tr>
<td>II</td>
<td>14 days</td>
<td>256.0±31.6</td>
<td>25.3±15.8</td>
<td>105.0±21.0</td>
</tr>
<tr>
<td>III</td>
<td>23 days</td>
<td>10.3±11.7</td>
<td>500.0±56.1</td>
<td>2352.0±36.2</td>
</tr>
</tbody>
</table>
Table 3. Changes in the blood coagulation indicators with rabbits under the influence of sound stimulation (N=3)

(1) Animal (2) Series; (3) Group; (4) Period of sound effect; (5) Indicator; (6) Blood coagulation time (in seconds); (7) Time of plasma recalcification (in sec); (8) Tolerance of the plasma to heparin (in sec); (9) Prothrombin complex; (10) fibrinogen (in mg%); (11) Studies; (12) Without effect; (13) 14 days; (14) 28 days; (15) Table 3 (continued); (16) XII factor (in %); (17) Adhesiveness of thrombocytes (in %); (18) Thrombin time (in sec); (19) Heparin time (in sec); (20) Fibrinolytic activity of the blood;

<table>
<thead>
<tr>
<th>Group</th>
<th>Indicator</th>
<th>Series</th>
<th>Period</th>
<th>Value (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>I</td>
<td>A</td>
<td>Without</td>
<td>12.3±7.4</td>
</tr>
<tr>
<td>II</td>
<td>II</td>
<td>B</td>
<td>Without</td>
<td>13.5±8.9</td>
</tr>
<tr>
<td>III</td>
<td>III</td>
<td>C</td>
<td>Without</td>
<td>14.7±9.2</td>
</tr>
</tbody>
</table>

(continued)
The material is fixed in 10% neutral formalin. After fixing of the aorta, everything is colored with sudan III. After composing the distribution schemes of the different sections of the aorta (arch, breast and abdominal segment), pieces from 5 to 10 are cut out (depending on the expression of the changes). The sections are prepared on a frozen microtome and colored oil red; the remaining part of the pieces were filled with paraffin-collodion, the sections were colored according to Van Gizon and according to Weigert-Chart on elastic fibers. The hearts of the rabbits of the experimental and control groups were cut into transverse segments (4-5) which were also studied like the aorta.

Experimental Results

The rabbits subjected to sound stimulation to the end of the experiment changed their behavior. They became aggressive, attacked each other, fought frequently. At the same time, they appeared frightened. At the sight of the experimenter, they hid in the corner of the chamber. With a large part of the rabbits subjected to the effect of sound in the course of 28 days, the weight dropped 600-1000 g. In the case of the rabbits subjected to the effect of sound for 16 days, the weight drop was less.

In the first series of experiments on rabbits on ordinary feed (group B), the sound stimulation did not cause an increase in the level of cholesterol and of the total fraction of β-lipoproteins; we noticed a real increase in the level of nonesterified fatty acids from 612.8±68.3 to 827.1±71.0 mg/l (p < 0.05). In the case of rabbits found on the same ration and not subjected to the effect of sound (group A), the changes in the level of nonesterified fatty acids were not observed in the study carried out at the same time. Under the influence of sound effect, there was an increase in blood coagulation (the recalcification time was shortened from 135.5±16.1 to 84.7±15.8 sec., p < 0.05). There was an increase in the tolerance of plasma to heparin from 254.7±32.5 to 135.8±24.5 sec (p < 0.05). The other hemocoagulation indicators did not substantially differ from the values of the control group (Table 2.3).

In opening up the rabbits of the different series of experiments, sacrificed at different times, we did not find any visible changes with a general examination. Visible changes did not appear in macroscopic studies of rabbit aorta of the 1st series, either with those subjected or not subjected to the effect of sound. With a microscopic study of the coronary arteries of the heart and myocardium of group B rabbits, we found changes in the form of hypertrophy of the intramuscular wall branches and sharply expressed venous hyperemia. Small focal points of necrosis were observed near the artery changes. Such changes on the side of the vessel wall and the myocardium were not observed in the case of the rabbits not subjected to sound effects (group A).
In the IIInd series of experiments with rabbits kept on ordinary food with the addition of cholesterol for a period of 4.5 months and "rest" after its removal during the last month, the level of cholesterol and of the total fraction of β-lipoproteids before the start of experiments was higher in both groups than in either group of the first series of experiments. A substantial difference was not found in the level of nonesterified fatty acids. With repeated studies for 14 days, the cholesterol level and the total fraction of β-lipoproteids were lower in both groups, which is connected with the cholesterol metabolism. The level of nonesterified fatty acids in group B increased from 607.5±70.9 to 797.5±69.1 mlekw/l (p < 0.05). The hemocoagulation indicators in the case of rabbits taken off cholesterol practically did not differ from their values in series I. Under the influence of stimulation (Table 2, 3), the tolerance of the plasma to heparin increased from 264.8±26.1 to 176.5±28.0 sec (p<0.05).

![Fig. 1. Irregular distribution of lipids in the aorta. x 106. Rabbit No. 473](image)

When opening up the rabbits of the IIInd series, the subcutaneous adipose layer in group B was expressed somewhat less than in group A. With microscopic examination of the aortas of the IIInd series of rabbits, we found atherosclerotic changes expressed in different degrees and they turned out to be alike in both groups. In groups B there were express changes on 3 of the 12, less clear ones on 5, slight ones on 4 of the rabbits. In group A, accordingly, on 3 of the 8, 3 and 2 of the rabbits (sic.). With microscopic examination in the atherosclerotic platelets* on part of the rabbits of group B we observed rather clearly expressed signs of lipid resorption in the form of unequal distribution of the latter in the platelets themselves, at places they were almost completely absent, the presence of a large number of cells with lipid inclusion in the cytoplasm (lipid macrophage) and the penetration of lipids in the middle membrane of the aorta (Fig. 1). Atherosclerotic changes, large

* This should be "plaques".
Atherosclerotic platelets were also observed in the coronary arteries of the heart and in the intermuscular and main branches of the coronary heart arteries. Also, in the aorta, in some platelets of the coronary arteries of the heart we observed an unequal distribution of the lipids, giving evidence of the start of their resorption (Fig. 2). Changes were found in the vessel wall and the myocardium, but more expressed, like in the case of the rabbits of group B of the 1st series. Considerable hypertrophy of the walls of many coronary arteries of the heart attracted attention (Fig. 3). Often we found considerable deposits of lipids in the thickened wall, not leading to the formation of platelets, however. As distinct from the intramuscular branches of the coronary arteries of the heart, in the main branches of the artery walls on the other hand we found that its lumen was widened. We turned our attention to the clearly expressed atherosclerotic damage in the coronary arteries of the heart (Fig. 4). In different sections of the myocardium, preferably close to the hypertrophied artery, we observed venous hyperemia, sometimes "old" blood effusion and very clear dystrophic changes (Fig. 5). The changes in the heart muscle appear as focal or diffuse fatty infiltration, although we must not exclude the possibility of its origin due to atherosclerotic heart damage. We also observed small focal points of necrosis and small network scars. In the case of the rabbits of group A, signs of lipid resorption in the aorta and in the coronary arteries were absent against a background of expressed atherosclerotic changes.

Fig. 2. — Large atherosclerotic platelets in the intermuscular branches of the left venal artery of a rabbit heart.

Above (a) — control, lipids with equal distribution in the platelets. × 300, rabbit No. 239; bottom (b) experimental— unequal distribution of lipids. ×116, rabbit no. 473.

* This should be "plaques".
Fig. 3.--Hypertrophy of the wall of the left coronary artery, edema. Above (a)--control x140, rabbit no. 495; below (b)--experimental x106. Rabbit no. 473.

In the case of the rabbits under the influence of extended maintenance on a cholesterol diet (Table 2), the level of cholesterol, of the total fraction of β-lipoproteins, nonesterified fatty acids was considerably higher than in the case of the rabbits of the 1st and 2nd series. For 2 (groups A and B) and 4 weeks (group B), the level of cholesterol, total fraction of β-lipoproteins with repeated study practically did not change. The level of nonesterified fatty acids in groups B and C dropped correspondingly from 1308.0±149.4 to 793.3±56.6 mEq/l (p<0.02) and from 1323±125.1 to 966.6±63.6 mEq/l (p < 0.01).
Under the influence of extended feeding with cholesterol in rabbits of the IIIrd series, the heparin and fibrinolytic activity in the blood turned out to be higher than in the I st series. In this regard, our data is closer to that observed by the other authors. [11] The applied sound stimulation facilitated an increase in the hypercoagulation properties of rabbits in groups B and C: the recalcification time was correspondingly shortened from 107.6 ± 12.1 to 61.8±9.8 sec (p < 0.05) and from 128.0±18.5 to 70.3±5.4 sec (p < 0.05), an increase of the adhesiveness of the thrombocytes from 20.7 ± 4.1 to 31.0 ± 2.4% (p < 0.05) and from 23.2 ± 3.0 to 36.4 ± 2.7% (p < 0.01), a decrease of blood heparin activity correspondingly from 12.8 ± 1.0 to 9.5 ± 1.2 sec (p < 0.05) and from 13.1 ± 1.1 to 8.5 ± 1.0 sec (p < 0.05), a decrease of fibrinolytic activity of the blood correspondingly from 5.0±1.1 to 1.83 ± 0.19 (p < 0.01) and from 5.9±1.63 to 1.75±0.24 (p < 0.05). In group V, in addition to these changes at the end of the sound effect, we noted a shortening of the blood coagulating time from 161.3±19.2 to 90.0±6.4 sec (p < 0.01), an increased tolerance of plasma to heparin from 168.2±18.2 to 104.6±6.1 sec (p < 0.05), an increase in the level of the XIIth factor from 95.4±15.2 to 148.5±17.2% (p < 0.05). It should be noted that heparin and fibrinolytic activity of the blood in rabbits of the IIInd series (groups B and C) at the end of the experiment did not exceed the values in the case of the rabbits of the I st series.

The morphological data with the macroscopic as well as with the microscopic studies turned out to be analogous with the IIInd series (in groups B and C with group B between groups A). However, the degree of expression of the atherosclerotic process turned out to be different in series III. So the atherosclerotic changes were slight in all 5 rabbits of group B which were studied morphologically. In the case of the rabbits of group B, the expressed atherosclerotic changes occurred in 2 of the 6, less expressed in 2 and weakly expressed in 2 of the rabbits. In group A, the expressed atherosclerotic changes were found in the case of 2, less expressed in the case of 1 and slightly expressed in the case of 3 rabbits. So, in the case of rabbits subjected to the effect of sound for a period of 28 days, the degree of atherosclerotic changes in the vessels was less expressed than in the case of rabbits with shorter stimulation.
Fig. 4.--Multiple damage to the intramuscular branches of the coronary arteries. Large constricted atherosclerotic platelets (x36, rabbit no. 505).

Fig. 5.--Clear degenerative changes in the myocardium, rabbit No. 499.

Above (a)--x 130; Below (b)--x 105.
Coloring oil red O + hematoxylin.
Discussion of the Results

As was seen, a prolonged acoustical effect exerted an influence on the changes of the animal level of nonesterified fatty acids and the indicators of the blood coagulating system. The level of nonesterified fatty acids in the experimental groups of animals changed unequally. An increase in the level of nonesterified fatty acids under the influence of the sound effect was observed with rabbits kept on ordinary food during the time of the study (I st series, group B), and in the case of the rabbits kept on ordinary feed for a period of 1.5 months (month up to the start of the experiment and during its time), but before this receiving a cholesterol diet for a period of 4.5 months (II nd series, group B). Drop in the level of nonesterified fatty acids was observed in groups of rabbits kept on a cholesterol diet for a period of 4 months up to the start of the experiment and during the sound effect (III rd series, groups B and C). The changes of the hemo
coa
gulation indicators were characterized by a general hyper
coagulation tendency, more expressed in the case of rabbits on a cholesterol diet during the time of the experiment (III series, groups B and C). The level of cholesterol and of the total fraction of β-lipoproteins did not substantially change under the influence of the sound effect.

Despite the different character of the humoral shifts or their absence under the influence of sound effects in series I, II, III, the morphological changes in the wall of the heart and myocardium artery turned out to be analogous—hypertrophy of the vessel wall, interruption of blood circulation in the myocardium (pithoric blood vessels), focal points of necrosis. Evidently, the latter is connected with an increase of the sympathetic-adrenal activity which facilitated changes of the me
tabolic processes in the vessel wall and in the heart muscle. The different character of the necrosis (from fresh to scar changes) indicates that they do not emerge simultaneously, but are connected with an extended, repeated sound effect.

The data obtained on animals kept on a diet with cholesterol and subjected to sound effect (series II and III) is especially interesting. The combination of a high level of lipids in the blood and hypercoagulation shifts in the hemostasis system, on the one hand, and the presence of lipid resorption on the platelets in the aorta and coronary arteries, on the other, attests to the complexity of the organism reaction, created under conditions of extended sound effect.
The results of the obtained data in a way contradict the prevailing data concerning the increased influence of the different types of stress on the degree of atherosclerotic changes in the experiment. This is especially confirmed by the degree of expression of the atherosclerotic changes observed in a group of rabbits subjected to more extensive sound irritation (article III, page 8). The reason for the fact observed by us has not yet been explained. Evidently, we may be dealing with a specific influence of the stimulation (sound) used by us. It is possible that this type of stimulation, when it is especially strong, caused special changes on the level of the hypothalamus in connection with the physical nature of the sound.

In the literature there is indication of the possibility of resorption of lipids from the platelets, created under certain conditions. One of the factors facilitating resorption of the lipids is an increase in the activity of the lipo-mobilizing factor of hypophysis whose released stimulation takes place under conditions of stress. According to the data of Б.М. Липовецково [6,7], with the introduction of the lipo-mobilizing factor of hypophysis into the rabbits with experimental atherosclerosis, resorption of the lipids takes place in the aorta and there is an increase of lipolytic activity of enzymes in the vessel wall. The indirect support of the high lipolytic activity of the vessel walls in our observations obviously may be considered to be a large number of macrophages observed in the platelets. According to the data of Зампланя и ал. [4], an increase of lipolysis in the vessel wall is connected with a great accumulation of macrophages in them, having a high enzyme activity. Possibly, a decrease in the level of non-saturated fatty acids is connected with an increase of the lipolytic activity [4, 13]. In our studies, the resorption of lipids from the platelets was accompanied by a decomposition of fat on the periphery.

So, in conditions of prolonged sound effect on animals with an expressed atherosclerotic process, despite the high level of lipids in the blood and the increased capability of the blood to coagulate, conditions are created facilitating the resorption of lipids from the platelets, against a background of the continued introduction of cholesterol. Additional studies are necessary to explain the mechanism of the obtained results.
Literature


[22] Sackatt, J. E.: J. biolog. chemistry, 64, 203, 1925.


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ФИЗИОЛОГИЧЕСКИЙ ЖУРНАЛ СССР им. И. М. СЕЧЕНОВА

ВЛИЯНИЕ ЗВУКОВОГО РАЗРЯЖЕНИЯ НА ЛИПИДНЫЙ ОБМЕН,
ПОКАЗАТЕЛИ СИСТЕМЫ СВЯЗИЯ КРОВИ И РАЗВИТИЕ
ЭКСПЕРИМЕНТАЛЬНОГО АТЕРОСКЛЕРОЗА У КРОЛИКОВ

Г. П. Деревянко, Т. С. Синицына, Т. В. Веселова

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Звуковое воздействие на организмы 14 дней их звуковых звуков способствует повышению уровня липидов и снижению вязкости крови. Морфологически выявлены гипертрофия и гиперплазия желудочков сердца, кровь в артериях и венцах. У крыс с экспериментальным атеросклерозом, подвергнутых звуковому воздействию и течением 12 и 24 недель, наблюдали значительное повышение уровня липидов в крови и коронарных сосудов. В группах с короноваскулярными заболеваниями было исследовано развитие липидов на ворота и коронарных артерий. Морфологически, кровь в артериях после звуков подвергнутых исследований, оказал повышение реактивности артерий и диффузным нарушениям интимы и меди, а также нарушениям стенок.

Многочисленными экспериментальными исследованиями установлено, что в процессе развития атеросклероза происходит утолщение стенок артерий, что приводит к нарушению кровотока и развитию атеросклеротических изменений в стенках артерий. Однако механизмы влияния звукового воздействия на развитие атеросклеротического процесса до сих пор не определены.

Целью данной работы стала оценка влияния звукового воздействия на развитие атеросклеротического процесса у крыс. МЕТОДИКА

Работа выполнена на 33 кроликах-самцах породы Нандиного весом 2,7—3,2 кг. Из 33 облученных крыс получены образцы в течение 5,5—6 мес. 500 кг лактозы в 5,0 широкополосного радиоугенератора, 15 крысам подвергали звуковому воздействию (га), 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га).

В остальных случаях использовали радиоугенератор (га) и 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га) и 5 — звуковому воздействию (га). Через 24 часа после введения холестерина 13 крысам из 20 были подвергнуты звуковому воздействию в течение 14 дней (группа 3). 8 крысам
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<th>Начало зацикливания</th>
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(группа А) не подвергалась зацикливанию, в III серии использовался 18 крысят, получавших холестерин в течение 4,5—5 месяцев, 12 из них через 4 месяца от начала нередкения холестерина были подвергнуты зацикливанию и в течение 14 дней — группа Б, в то время как группа А подвергалась зацикливанию через 4 месяца. Затем крысята были разделены на группы, которые подвергались зацикливанию (группа А), и группы, которые не подвергались зацикливанию (группа Б).

Описанная методика эксперимента была использована для исследования влияния зацикливания на уровень холестерина. Группа А подвергалась зацикливанию с помощью лиофилизированного зацикливателя, который был введен в их организм через 4 месяца после начала эксперимента. Уровень холестерина в крови крысят снизился с 632,5 ± 38,3 до 177,2 ± 17,1 мг/л (р < 0,05). У крысят группы Б, не подвергавшихся зацикливанию, уровень холестерина остался на высоком уровне. В результате проведенных исследований было установлено, что зацикливание снижает уровень холестерина в крови крысят, что может быть использовано в качестве метода лечения холестериновой гиперхолестеринемии. Результаты исследования свидетельствуют о том, что зацикливание может быть эффективным методом лечения холестериновой гиперхолестеринемии. В дальнейшем планируется проделать дополнительные исследования для подтверждения полученных результатов.
Возрастание концентрации (мкмл.) холестерина у крыс после повреждения аorta
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При использовании различных серий опытов, убитых в разные сроки, видимых изменений при общем осмотре не обнаружено было. При микроскопическом исследовании в кюветах I серии, как подперероганищих, так и не задержавшихся, зону кровоизлияния, видимых изменений не вызывали. При микроскопическом исследовании венечных ветвей сердца и миокарда у крыс в группах В обнаружены изменения в виде гипертрофии стенок эндокардиальных ветвей и неровности поверхности гипертрофированной. Вследствие отмеченных авторами наблюдений были взяты образцы. У крыс, не подвергавшихся зону кровоизлияния, при изучении структуры сосудистой стенки и миокарда не было. 

В кювет I серии опытов у крыс, подвергавшихся неразличимым изменениям в тканях исследуемого органа, уровни холестерина и суммарной фракции 2-липопротеинов перед началом опыта в обеих группах были выше, чем в опытных группах. Уровни 

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<td>28 дней после повреждения</td>
<td>7,3 ± 1,1</td>
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При использовании различных серий опытов, убитых в разные сроки, видимых изменений при общем осмотре не обнаружено было. При микроскопическом исследовании в кюветах I серии, как подперероганищих, так и не задержавшихся, зону кровоизлияния, видимых изменений не вызывали. При микроскопическом исследовании венечных ветвей сердца и миокарда у крыс в группах В обнаружены изменения в виде гипертрофии стенок эндокардиальных ветвей и неровности поверхности гипертрофированной. Вследствие отмеченных авторами наблюдений были взяты образцы. У крыс, не подвергавшихся зону кровоизлияния, при изучении структуры сосудистой стенки и миокарда не было. 

В кювет I серии опытов у крыс, подвергавшихся неразличимым изменениям в тканях исследуемого органа, уровни холестерина и суммарной фракции 2-липопротеинов перед началом опыта в обеих группах были выше, чем в опытных группах. Уровни 

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При повторном исследовании через 14 дней уровень холестерина и суммарной фракции 2-липопротеинов в обеих группах был выше, чем в контрольных. Уровень 

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капилляра гемоагуляция у кроликов после отмены холестерина практически не отличалась от значений их в I серии. Под влиянием раздражений (табл. 3-7) наблюдалась телоплазмальная абляция и генерация с 264,8±29,1 до 178,3±23,0 сл. (p < 0,05).

При вскрытии кроликов I серии подкожный жировой слой в группе В был выражен несколько меньше, чем в группе А. При микроскопическом исследовании сердца I серии кроликов были обнаружены атеросклеротические изменения, выраженные в различной степени, и в обеих группах они оказались одинаковыми. В группе В разница в размере у 5, слабо у 4 кроликов; в группе А соответственно у 3 на 8,5 и у 2 кроликов. При микроскопическом исследовании в атеросклеротических бляшках у части кроликов группы В наблюдались довольно

Рис. 1. Неравномерное распределение липидов в аорте.

отчетливо выраженные признаки резорбции липидов в виде неравномерного распределения последних в стенке, почти полное их отсутствие, наличие большого количества клеток с липидными включе-

ними в цитоплазме (липидные микрофиляры) и прожилками липидов в сред-

нюю оболочку аорты (рис. 1). В венах артериальных сосудов также наблю-}

даемых ассоциаций изменение, крепких атеросклеротических бляшек во внутренних ветвях и главных ветвях венечных артерий сердца. Там же как и в аорте, в некоторых ветвях коронарных артерий сердца наблюдалось неравномерное распределение липидов, свидетельствовав-}

ше о начале их резорбции (рис. 2). В сосудистой стенке и мышце сердца были обнаружены изменения, казавшиеся неровными, как у кроликов группы В I серии. Обнаруживая на себя внимание значительных гипертрофии стенки внутренних артерий сердца (рис. 3). Период в утолщенной стенке определялись значительные отложения липидов, не приводя, однако, к образованию бляшек. В отличие от внутренних ветвей и главных ветвей венечных артерий сердца, в обеих группах кроликов, наблюдалось наличие большого количества атеросклеротических поражений в коронарных артериях сердца (рис. 4). В различных деталях мышцы, преимущество волокон от гипертрофированных артерий, наблюдалась волокнистая гипертрофия, иногда «стяжка» кровеносных и очень резко деструктивных изменений (рис. 5). Начавшие в сердечной массе представляли собой очаговую или диффузную инфильтрацию, хотя нельзя исключить, что и волокнистые волокна повышенном в счет атеросклеротических поражений сердца. Наблюдались такие инфарктные очаги небольшого и небольшие от-
чтые рубцы. У кроликов группы A на фоне выраженных атеросклеротических изменений в артериях отсутствовали.

У кроликов III группы под влиянием длительного применения холестерина и фибринолитика НЭЖКК был значительно выше, чем у кроликов I и II серий. Через 2 недели (группа B) уровень холестерина, суммарной фракции β-липопротеинов в группах B и II значительно снизился соответственно с 1305,0 ± 149,4 до 733,3 ± 55,6 мг% с/л (p < 0,02) и с 1335,1 ± 125,1 до 396,8 ± 63,0 мг% с/л (p < 0,01).

При длительном применении холестерина у кроликов III серии гепатомегалия и фибринолитическая активность крови оказалась выше, чем в I серии. Параметры в этом отношении были схожими с наблюдавшими другими авторами [11]. Применение экзогенного холестерина способствовало увеличению гиперпротеинемических свойств крови в группах B и II:
укорочение времени рекапилляции соответственно со 107,6±12,1 до 61,8±5,8 сек. (р < 0,05) и со 128,0±18,5 до 70,3±5,4 сек. (р < 0,05), незначительно сокращение тромбинон времени соответствовало 30,7±4,1 до 31,0±2,4 сек. (р < 0,05) и 23,2±3,0 до 26,4±2,7 сек. (р < 0,01), снижение гепариновой активности крови соответственно с 12,8±1,0 до 9,5±1,3 сек.

Рис. 3. Гемодиагностическая схема ленты изображения артериальной системы.

В центрограммах (а) — контроль, ×100, площадь 24499; в центрограммах (б) — опыт, ×100, площадь 2472.

(р < 0,05) и с 13,1±1,1 до 8,5±1,0 сек. (р < 0,05), снижение фибринолитической активности крови соответственно с 5,8±1,1 до 1,8±0,1 сек. (р < 0,01) и с 5,9±1,0 до 1,75±0,24 сек. (р < 0,05). В группе В, кроме этих изменений, к концу зондового воздействия отмечалось укорочение времени свертывания крови со 161,3±10,2 до 90,0±6,4 сек. (р < 0,01), повышение гемостаза и активность генерации крови 104,8±6,1 сек. (р < 0,05), повышение уровня XIII фактора с 95,4±15,2 до 148,5±17,2% (р < 0,05). Необходимо отметить, что генерация и фибринолитическая активность крови у крыс после инъекции III серии (группы В и III) в конце опыта не показала значений снижения у крыс после I серии.
Морфологические данные как при микроскопическом, так и при микроскопическом исследовании оказались сходными со II сериями (в группах Б и В с группами Б и группами А). Однако степень выраженности атеросклеротического процесса оказалась разной в III серии. Так,

у всех 6 кроликов группы В, обследованных морфологически, атеросклеротические изменения были слабыми. У кроликов группы Б выраженные атеросклеротические изменения были у 2 из 6, менее выраженные у 2 и слабые у 2 кроликов. В группе А выраженные атеросклеротические изменения выявлены у 2, менее выраженные у 1 и слабые у 3 кроликов. Таким образом, у кроликов, подвергавшихся звуковому воздействию в течение 28 дней, степень атеросклеротических изменений в сосудах была менее выражена, чем у кроликов с более коротким раздражением.

ОБСУЖДЕНИЕ РЕЗУЛЬТАТОВ

Дополнительное звуковое воздействие, как было видно, оказывало влияние на изучение у животных уровня НЭЭКК и показателей системы свертывания крови. Уровень НЭЭКК в опытных группах животных изменился необычно. Повышение уровня НЭЭКК под влиянием звукового воздействия наблюдалось у кроликов, содержавшихся во время исследований на обычном корме (I серия, группа Б), и у кроликов, содержащихся на обычном корме в течение 1,5 месяцев (месяц до начала опыта и во время ного), но в более значительном масштабе. Уровень НЭЭКК наблюдался в группах кроликов, содержащихся на холестериновой диете в течение 4 месяцев до начала опыта и в течение звукового воздействия (III серии, группы Б и В). Изменения на показателях гемоглобин-лизиновой реакции, более выраженной у кроликов, находящихся во время исследований на холестериновой диете (II серии, группы Б и В), несколько отличались от направленного влияния звукового воздействия.

Несмотря на реальный характер гуморальных сдвигов или их отсутствие под влиянием звукового воздействия в I, II, III сериях, морфологического изменения в сосудах не наблюдалось. В III серии, где кроликов подвергали звуковому воздействию в течение 28 дней, степени атеросклеротических изменений в сосудах была менее выражена, чем у кроликов с более коротким раздражением.
EFFECT OF THE SOUND STIMULATION ON LIPID METABOLISM,
PARAMETERS OF THE BLOOD COAGULATING SYSTEM
AND DEVELOPMENT OF EXPERIMENTAL ATHEROSCLEROSIS
IN RABBITS

G. P. Deriagina, T. S. Sintilina and T. V. Vasileva


The 14-day sound stimulation of healthy rabbits increases the level of unsaturated fatty acids and the blood coagulability. Morphologically the hypertrophy of the heart coronary arteries and necrotic foci in the myocardium are revealed. In rabbits with experimental atherosclerosis subjected to sound stimulation during 14 and 28 days, in spite of a high level of lipids in the blood and hypercoagulatory shifts, the developing conditions aid in absorption of lipids from aortical plaques and coronary arteries. Apart from that, morphological studies reveal hypertrophy and occlusion of the vascular wall, necrotic foci, local and diffuse fatty infiltration of the myocardium.
гические изменения в сосудистой стенке артерий сердца и миокарда оказались сходными — гипертрофия сосудистой стенки, нарушение кровообращения в миокарде (полиморфное кровоизлияние сосудов), очаги некроза.

Последнее, очевидно, связано с повышенным симпато-адреналиновым активством, что способствовало нарушения метаболических процессов в сосудистой стенке и в сердечной мышце. Разный характер некрозов (от слабых до грубоватых) говорит о том, что они возникают не одновременно, а сопровождаются длительными, повторяющимися звуковыми воздействиями.

Собрано интересные данные у животных, содержавшихся на диете с холестерином и подвергавшихся звуковому воздействию (II и III серии). Сочетание высокого уровня липидов в крови и гипергликемии животных также в системе гомеостаза, с одной стороны, и наличие разорванных артерий в аорте и коронарных артериях — с другой, говорит о сложности развития органов, живущих в условиях длительного звукового воздействия.
Результаты полученных наблюдений касаются противоречий и chezших мнений об успокаивающем влиянии различных средств стресса на структуру часто варировающих изменений в эксперименте. Особого внимания заслуживают меньшие изменения вестибулярных изменений, обнаруженных в группах контроля, подвергшихся более длительной шумной вибрации (III серия, группа В). Причем наблюдаемые изменения на уровне гипоталамуса в связи с физическими напряжениями.

В литературе существуют указания на возможность разложения диапазонов в условиях, создающихся в определенных условиях. Одним из факторов, способствующих разложении диапазонов, является повышенная активность ликвотворящего фактора гипоталамуса, стимулирующего выделение норадреналина в условиях стресса. По данным В. М. Липовецкого [4, 7], при введении ликвотворящего фактора гипоталамуса происходит разложение диапазонов вибрации и уровня и показателя ликвотворящей активности в условиях стимуляции. Коэффициенты корреляции низкой ликвотворной активности сусудистой стенки и высокой вибрации, очевидно, можно считать большие, но эти факторы, обладающие сложной структурой и высоким уровнем активности, могут быть объяснены более сложными причинами. В наших исследованиях разложение диапазонов вибрации связано с повышением ликвотворной активности вибрации и уровня активности НОЖК [4, 7]. В наших исследованиях разложение диапазонов в условиях стимуляции и аспекта уровня НОЖК.

Таким образом, в условиях длительного шумового воздействия у животных с выраженным вегетативным стрессом, отмечен на высокой активности вибрации и повышенной активности нервной системы, способы разложения диапазонов вибрации на фоне продолжающегося введения нейролептик. Необходимы дальнейшие исследования для выяснения механизма полученных результатов.

**Литература**

**SUMMARY FORM FOR**

**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

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<tr>
<th>Principal Investigator(s)</th>
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<tr>
<td>L. Verdun di Cantogno</td>
<td>Dept. of Audiology and 1st Dept. of Medical Pathology, University of Turin, Turin, Italy</td>
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<td>R. Ballarba</td>
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<td>F. S. Tonagga</td>
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<td>L. Coiola</td>
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**Investigator's Phone No.**

**Sponsoring Organization**

University of Turin


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**Type & duration of experiment:**

Type: Controlled laboratory experiment in an Amplifon Standard G5 silent booth

Duration: 105 min. per subject

**Purpose for study:**

To study the effects of 10 min. of traffic noise on the cardiovascular system, blood chemistry and urine levels of catecholamines

**Description of test groups (subject, e.g., age, etc.):**

33 male subjects aged 20 - 70 years divided into 3 test groups (which were divided into subgroups of 20 - 45 yr. olds and 46 - 70 yr. olds):

1) 11 normals (average age 36.7 yrs.)
2) 11 diabetic or with abnormal fat levels (average age 46.4 yrs.)
3) 11 control group - 11 normal male subjects (average age 27 yrs.)

**Statistical Methods:**

Students t-test

**Control of other regressors:**

Laboratory conditions used; blood samples taken using butterfly needle to avoid stress from procedure

**Noise stimulus:**

Source: Taped traffic noise from 10 areas of Turin

Spectral characteristics: not given

Noise level: mean Leq=71.6dB; 88.8dB

Length of exposure: 10 min.

**# of trials:**

1 per subject

15 min. quiet - 10 min. traffic noise - 60 min. quiet

**CVS Response Measured:**

1) blood pressure 2) heart rate

**Non-auditory effects:**

CVS: Blood pressure and heart rate increased due to traffic noise; electrocardiograms - no significant changes due to noise

**Other:** Noise stimulated increases in urinary catecholamines, blood sugar, insulin, cholesterol, urine sodium

**Author's conclusions:**

Traffic noise caused increased blood pressure and heart rate, and altered the blood chemistry such that this type of noise stress may be a coronary risk factor and may be involved in the development of arteriosclerosis. Blood chemistry data was too small to make definite conclusions, however.

**Evaluation & comments:**

1) no age-matched control group - mean age of controls is 10 years less than the youngest test group. 2) 10 min. noise stimulus - too short from which to draw any real conclusions.
The effects of traffic noise on blood pressure, heart rate, electrocardiogram, blood chemistry, and urinary catecholamines were studied in 33 male subjects, aged 20 to 70 years. The subjects were divided into 3 groups as follows: 11 healthy subjects (average age 36.7 years); 11 with heart disease (average age 52 years); 11 having diabetes or abnormal lipid metabolism (average age 46.4 years). A control group of 11 healthy males (average age 27 years) was subjected to all of the same procedures as the 33 test subjects, except traffic noise. Both tests and controls had normal hearing for their ages. After a 6 hour fast, the subjects were placed on a bed in an Ampliﬁon standard GS silent booth. Following 15 minutes of quiet, a 10-minute tape recording of traffic noise from 10 areas of Turin, Italy was introduced into the subjects’ headphones. The mean noise level was 71.6dBA, and the Leq for 10 min was 73.1dBA. The subjects remained in the test chamber under quiet conditions for 80 minutes after the tape ended. Blood samples were taken 20 minutes before and immediately before the noise stimulus—the average of the 2 sets of results was used as the baseline value for each blood chemistry test. Blood samples were also taken when the tape stopped, 20 minutes later, and 50 minutes later. The blood pressures and electrocardiograms were measured before, during, and after the noise stimulus. Urinary catecholamines were measured on samples taken before and after the traffic noise. The students t-test was used to analyze the data obtained both between groups and to compare the results within groups due to age. Traffic noise stimulated catecholamine excretion in the test group with diabetes or abnormal lipid metabolism. The blood samples were analyzed for sugar, uric acid, total lipids, cholesterol, triglycerides, and insulin. Changes in blood chemistry due to noise were observed in all 3 test groups. The greatest changes occurred in the group with abnormal metabolisms. Noise induced increased blood sugar and uric acid levels, indicating that noise affects nucleic acid metabolism. Total lipid levels were the same as that of the controls in the healthy test group; total lipids increased in the other 2 test groups. Triglyceride levels increased due to noise, especially in subjects over 45 years. Cholesterol levels increased due to noise, especially in the heart patients. Insulin levels increased due to noise in the healthy subjects and in those with abnormal metabolism, whereas insulin levels decreased in the heart patients. No significant changes were found in the electrocardiograms due to noise. The heart rates and systolic blood pressures increased due to noise in all subjects. The results indicate that traffic noise may be a risk factor in the development of arterial and coronary disease.
URBAN TRAFFIC NOISE, CARDIOCIRCULATORY ACTIVITY AND CORONARY RISK FACTORS

L. Verdun di Cantuagra, R. Dallerba, P.S. Teagno, L. Cooole
From the Department of Audiology and the 1st Department of Medical Pathology, University of Turin, Turin, Italy

SCOPE OF THE RESEARCH

The aim of this research was an assessment of the effect of road noise lasting 100 on cardiovacular activity and various blood chemistry indices, particularly those apparently associated with the pathogenesis of atherosclerosis. Normal, dysmetabolic and coronary subjects were examined.

While it can readily be appreciated that certain stimuli can act on the cardiovascular apparatus, leading to changes in the work of the heart and its performances, it is still to understand how certain psychological and emotional conditions, or certain sensorial stimuli, are completely mediated by the CNS, influence the heart and its vessels, thus supporting atheroma, cardiovascular thrombosis or serious arrhythmias.

Acoustical stimuli offer an ideal method for the study of effects mediated by the CNS, since they can be exactly reproduced and measured in the presence of every other factor likely to influence the frequency and arterial pressure, or left particular to a particular performance as assessed by simultaneous ECG, phonocardiographic and carotid recordings. Attention was also given to blood chemistry indices known as related coronary risk factors. Direct evaluation of cholesterol levels was not attempted, because the technique gives widely scattered results, even for normal conditions. Urinary catecholamines excreted during the test were however taken into consideration. The limitations of the method are well known.

MATERIAL AND METHODS

The absence of literature data concerning the effect of traffic noise on the parameters chosen for study suggested the advisability of preceding the experiment by a series of investigations of the effect of white noise and speech noise (100 db and 80 db intensity; continuous stimulation for 10' or for 1' followed by an interrupted stimulation for 5') in 16 subjects, to determine the effect of noise of different spectrum and intensity administered with different modalities. This preliminary study (details in the press) showed statistically significant differences between stimulated and control subjects. We therefore began by exposing 33 subjects aged 20-70 yr. to road noise. A further 11 normal subjects (mean age 27 yr) were studied for comparison, i.e. they were subjected to all the experimental procedures in the absence of road noise (control group).

Prior audiometric examination showed that all subjects had normal hearing for age. Only male subjects were examined, so as to ensure that responses were not affected by neurorhinal and neurorhynic factors pertinent to the two sexes.

The patients were divided into 3 groups (11 normals, average age 36.7 yr; 11 diabetic or dyslipidemic, average age 46.4 yr; 11 coronary, average age 45 yr). As already stated, the average age of the 11 controls was 27 yr. A division was also drawn between subjects aged more and less than 45 yr., i.e. 1' mean
57.3 yr.) and 16 (mean 32.7 yr.) respectively, excluding the controls.
Fifteen minutes after being settled on a bed in an Amplifier standard Q5 silent booth erected in a room lined with soundproof panels, each subject was exposed to a tape consisting of ten one-minute recordings of traffic noise registered at 10 different points in Turin. Its characteristics were:

<table>
<thead>
<tr>
<th>L_m dB</th>
<th>L_m dB</th>
<th>dB</th>
<th>L_m dB (A)</th>
<th>L_m dB (A)</th>
<th>dB</th>
</tr>
</thead>
<tbody>
<tr>
<td>88.8</td>
<td>89.4</td>
<td>2.55</td>
<td>71.6</td>
<td>72.1</td>
<td>0.54</td>
</tr>
</tbody>
</table>

This noise was fed through a Uher 4400 Stereo Record I C recorder to the circuit of an Amplaid 500 Audiometer calibrated in dB SPL, and then to the subject's headphones. It was used at its true intensity, which was checked with a Krol & Kjear photometer fitted with a model 1613 octave filter.

Neither the normal nor the control subjects displayed basal changes in blood chemistry parameters, in blood pressure or in electrocardiographic and phonocardiographic patterns. Each patient had been fasting for at least 6 hr. Five blood samples were taken as follows: 20' and immediately before stimulation; the arithmetic mean of these values was used as the "basal value" — 10', when the tape stopped, and 30' and 90' after it started. To avoid problems of stress related to the blood samples a butterfly needle was used, kept pat ent with saline.

There was no significant difference between the two basal values, bearing in mind the scatter displayed by these data under physiological conditions: Sugar, insulin, uric acid, total lipids, cholesterol and triglycerides were measured in each sample, using the following methods:

- **Blood Sugar:** Enzymatic determination with G6-PD-hexokinase (Biochemical test combination kit).
- **Blood Insulin:** Richter radioimmunological kit, utilizing a "Packard" Triarch Liquid Scintillation Spectrometer.
- **Blood Uric Acid:** "Uricum Quant" colorimetric enzymatic method (Biochemical Test Combination).

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**Total Lipids:** Colorimetric method of Zeichiner & Kirsch.
**Cholesterol:** Biochemistry Test Combination Kit according to D. Watson, B. Zuk and H.H. Lefler.
**Triglycerides:** Enzymatic determination of serum concentration with Biochemistry Test Combination Kit.

Urinary catecholamines were determined fluorometrically immediately before and immediately after the examination, so that their excretion during the test could be known by their concentration in the samples.

Blood pressure was measured before, during and after the stimulation with road noise 0-1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16-20, 20-40, 40-60, 60-90' with an Erka "Diastat" apparatus, using a pneumatic cuff fitted with a microphone for the registration of Koroskoff tones. Systolic and diastolic pressure values were automatically indicated every minutes on charts with a margin of error of less than 1.5%.

ECG and polygraphic data were obtained with a 8-channel Elema Schonander "Mingagraf II", giving a simultaneous ECG, 4-frequency phonocardiogram and carotid pulse recording. Polygraphic records were taken at the same intervals as those used to record arterial pressure. Examination of the ECG, phonocardiogram and carotid pulse data gave and induction, inter alia, of cardiac frequency. The distance, in terms of time, and the relation between certain features of the heart cycle shown by these recording were then calculated, so that the influence of the traffic noise on the heart performance could be detected and studied.

The following systolic times and indices were considered:

- **LVET (left ventricular ejection time):** from the root of the ascending branch of the carotid sphygmonogram to its dicrotic inflexure.
- **S1S2 (mechanical systole):** from the 1st component of the 1st sound to the aortic component of the 2nd sound.
- **PP (pre-ejection period, or tension-time, or pre-ejection systole):** from the start of the Q wave to the root of the ascending branch of the carotid sphygmonogram, less the PTT.
- **PTT (pulse transmission time):** from the

The initial values, applying Student P = 0.05. The each of the trials series as a whole between subjects.

After illustrate parameter separability of their behavior coronarypathic.

**BLOOD C**

Sugar.

Road noise added blood sugar in 50% of cases patients (Fig. in 50% of the they were significantly.)

(Sug. Fig. 2.)
nastic component of the 2nd sound to the
dicrotic incisure of the carotid pulse.
— Q1-S1 (Total or electromechanical systole):
from the start of the Q wave to the first com-
ponent of the 1st sound.
— Q3-S1 (deformation time): from the start of
the Q wave to the first component of the 1st
sound.
— ICT (isovolumetric contraction time): from
the first component of the 1st sound to the foot
of the ascending branch of the carotid sphyg-
nomogram, less the PTT.
— PEP/LVET expresses left ventricular per-
formance. LVET/ICT seems more closely rel-
lated to myocardial contractility.

The arithmetical mean of 10 beats was cal-
culated for each systolic time corrected for
frequency and compared with the normal theo-
retical values, to obtain significant data on
myocardial performance. In addition, the pro-
duct of cardiac frequency with systolic arterial
pressure offers a good indication of coronary
flow and myocardial oxygen needs.

RESULTS
The data were expressed as percent of the
initial values referred as 100, and analyzed by
applying Student's interval estimation, with
P = 0.05. The controls were compared with
each of the three groups and with the stimulated
series as a whole. Comparison was also made
between subjects aged less and more than 45 yr.
After illustrating the pattern displayed by each
parameter separately, an account will be given
of their behaviour in normal, dysmetabolic and
coronaropathic subjects.

BLOOD CHEMISTRY PARAMETERS

Sugar.
Read noise brought about an immediate increase
in blood sugar. This was more evident (over
50% of cases) in coronary and dysmetabolic
patients (Fig. 1). At 30' values were still high
in 30% of the normal subjects probably because
they were younger. Values, in fact, differed
significantly in function of age at 30' and 90'
(Fig. 2).

Insulin.
Traffic noise was accompanied by a marked
increase in band width in both normal and
dysmetabolic subjects. This was more evident
at 90', especially in the normal cases (Fig. 3).
At the end of the test, levels were well up in
about 80% of normal and 50% of dysmetabolic
subjects, whereas they were down in 50% of
the coronary group. This, once again, may be
attributed to age, since a marked, late rise in
blood insulin is typical in younger subjects
(Fig. 4).


Serum cholesterol. Subjects aged between 20 and 48 yr. Subjects aged between 48 and 70 yr.

Triglycerides.
Rapid rise caused a fall in values that appeared earlier (10' and 30') in the dysmetabolic as opposed to the coronary subjects. At 90' there was an increase of band amplitude in the normal group (Fig. 6). The fall at 30' was more marked in patients over 45 yr. (Fig. 7).

Blood cholesterol.
Values increased after 30' and 90' in about 50% of the normal subjects. Increased band amplitude was noted from the start, especially in the coronary patients (Fig. 8). There was no significant difference in function of age.

Uric acid.
A marked increase was observed at 30' and 90', especially in the dysmetabolic group (Fig. 9). Band amplitude also increased at an early stage in the coronary subjects, and later in the normal group. Up to 30', increases were higher in the younger subjects (Fig. 10).
Fig. 6. Triglycerides. Normal subjects ——; Dysmetabolic subjects ——: Coronary patients O —— O. Range of variation in unstimulated subjects. Range of variation in stimulated subjects.

Fig. 8. Blood cholesterol. Normal subjects ——; Dysmetabolic subjects ——: Coronary patients O —— O. Range of variation in unstimulated subjects. Range of variation in stimulated subjects.

Fig. 7. Triglycerides. Subjects aged between 20 and 45 yr, O —— O. Subjects aged between 46 and 70 yr, O —— O.

Fig. 9. Iron stores. Normal subjects ——; Dysmetabolic subjects ——: Coronary patients O —— O. Range of variation in unstimulated subjects. Range of variation in stimulated subjects.

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Catecholamines.

Values observed before and after the test are shown in Table 1. To obtain more accurate data it would have been necessary to keep the subjects completely at rest for 24 hr. before and after the test. This, however, was not possible. In spite of their limitation, the data showed that road noise enhanced catecholamine excretion in the dysmetabolic group. The relatively slight entity of the stimulus and its nature lead one to suppose that nothing less than direct haematocional determination of the catecholamine content would have given an accurate picture of this parameter. The techniques required, however, are difficult to apply in vivo, since very low quantities are involved. Furthermore, catecholamines are very rapidly inactivated in the body.

ECG AND HEART PERFORMANCE

Road noise did not lead to any significant ECG changes, even in subjects with marked basal signs of chronic or sub-chronic myocardial ischemia. During the course of the experiment isolated atrial or ventricular extrasystole was observed in some subjects, though these appeared to be devoid of particular significance.

Left ventricular performance was indirectly assessed polygraphically by simultaneous ECG, phonocardiographic and carotid pulse recording. Comparison was made between the length of the PEP and LVET, and of the relative importance on the ICT, with respect to the initial DT. The reciprocal relation between these parameters are indirect pointers to heart performance, especially as far as possible ischemia of the cardiac fibers is concerned. Our results did not reveal any marked variations in these and in other parameters. The noise used was apparently insufficient to influence these indices to an appreciable extent.

Changes in cardiac frequency were considered separately and together with systolic arterial pressure. As already stated, frequency times systolic pressure is regarded as one of the best pointers of coronary flow, tension time index and myocardial oxygen consumption. Traffic noise was responsible for a distinct increase of this index in the normal subjects, followed by a fall in response during the application of the stress (Fig. II A). A somewhat similar pattern was noted in the dysmetabolic group, though here values remained significantly high in 50% of cases until the end of the noise, after which there was a certain widening of the band (Fig. II B). In the coronary patients the increase was significant for a longer period in a larger number of cases (Fig. II C).

CONCLUSIONS

Exposure to road noise in the form employed in our experiment was followed by enhancement or depression of several blood chemistry parameters, or a wider scattering of their values, as shown by an increase of band amplitude around a more or less unchanged mean. On many occasions both responses were present, though their relative importance varied. Blood sugar displayed the earliest changes by contrast with the late response observed in the case of blood insulin.

We were particularly struck by the extent of the blood uric acid response, showing that noise has an effect on the metabolism of nucleic acids. As was to be expected, normal subjects generally presented less significant changes, especially in total lipids and triglycerides. An immediate increase in blood sugar was noted only in 50% of this group, as opposed to 80% of the other's.

Fig. 10. Blood uric. Acid. Subjects aged between 20 and 45 y. \( \frac{1}{2} \) subjects aged between 46 and 70 y.
Fig. 11. Cardiac frequency times systolic arterial pressure: a) Normal subjects ————, b) Dysmetabolic subjects ————, c) Coronary patients ————, d) All, b), c); Unstimulated subjects , , , , , , ,
Table 1. Catecholamines extracted by patients before and after the test, values in pg/ml

<table>
<thead>
<tr>
<th>Subjects</th>
<th>First week</th>
<th>Second week</th>
<th>Mean</th>
<th>sd</th>
<th>T value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretest</td>
<td>10.0 ± 2.0</td>
<td>12.0 ± 3.0</td>
<td>11.0</td>
<td>2.2</td>
<td>1.5</td>
</tr>
<tr>
<td>Posttest</td>
<td>15.0 ± 4.0</td>
<td>18.0 ± 5.0</td>
<td>16.0</td>
<td>4.0</td>
<td>2.5</td>
</tr>
</tbody>
</table>

Note: These samples were obtained from only 5 of the 10 patients.
whereas the late insulin response was more marked and frequent. This was the only group to display a late increase in cholesterol values, while its changes in uricemia included both decreases and increases, leading to an over 100% enlargement in band amplitude at 90°.

Diabetic and dyslipaemic subjects presented the most significant changes in total lipids, triglycerides and uric acid. This group displayed the most evident and earliest (10° and 30°) fall in triglycerides, by contrast with the increase in total lipids and their wider band amplitude at 30° and 90°.

Coronary patients showed a similar pattern, though apart from blood sugar, their changes were less striking. In this group the main features were variations in band amplitude, except in the case of total lipids, whereas triglycerides values shifted less than in the dysmetabolic subjects, but earlier than in the normal group.

The product of cardiac frequency and systolic arterial pressure — an index of change in coronary flow and the metabolic requirements of the myocardium — increased to a greater extent and for longer after the cessation of the road noise in the coronary patients, as was to be expected on theoretical grounds. This index tended to fall during the stimulation period in the normal subjects, whereas it stayed high throughout this period in the dysmetabolic group. This behaviour shows that exposure to traffic noise may be responsible for an increase in myocardial energy requirements by influencing frequency and systolic pressure.

Our blood chemistry data are to scanty to permit full interpretation of the changes observed. It can, however, be stated that road noise of the type employed can lead to distinct changes in all the parameters taken into consideration, these being of great importance as coronary risk factors in general terms, and as tending to support atheroma.
<table>
<thead>
<tr>
<th>Principal Investigator(s):</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. Drottn</td>
<td>Dept. of Audiology, Otolaryngology, and Internal Medicine, University Hospital, S-750 14 Uppsala 14 Sweden</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Investigator's Phone No.</th>
<th>Sponsoring Organization</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dept. of Audiology, Otolaryngology and Internal Medicine</td>
</tr>
</tbody>
</table>

**Citation:** Drottn, B. et al. Cardiovascular risk factors and hearing loss. A study of 1000 fifty year old men. Acta Otolaryngol. 79: 366-371, 1975

<table>
<thead>
<tr>
<th>Type &amp; duration of experiment</th>
<th>Purpose for study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type: Health examinations performed on subjects and medical histories were recorded.</td>
<td>To determine if there is a correlation between cardiovascular risk factors and hearing loss (sensorineural)</td>
</tr>
</tbody>
</table>

**Description of test groups (subject, age, sex, etc.):**

- 1000 50 year old men in 3 socio-economic levels: 1) professionals and other highly educated people, 2) school teachers at elementary level, clerical workers, small businessmen, 3) blue-collar workers, salaried craftsmen and service workers.

**Control of other variables:**

- No controls - smoking habits of the subjects were considered.

**Statistical Methods:**

- Chi-square test and the Kolmogorov-Smirnov test (for noise exp. and smoking habits)

**Noise Stimulus:**

- Noise exposure (qualitative) histories of the subjects were recorded; socio-economic level 3 had greater occupational noise exposure and hearing loss (most of the subjects in the other 2 levels had been in the military and could have some noise exposure).

**Non-auditory effects:**

- CVS, see Author's conclusions

**Author's conclusions:**

- No significant correlations between cardiovascular risk factors and hearing loss were found. Further study is needed.

**Evaluation & comments:**

- The three groups of subjects contain too many different occupations lumped together. Actual noise exposures of the subjects were too vague to make definite conclusions. The authors were not directly interested in the effects of noise on the cardiovascular system.

A health survey of 1000 healthy 50 year old men in Uppsala, Sweden was done to determine if a correlation exists between hearing loss (sensorineural) and various cardiovascular risk factors. The men were divided into 3 groups, according to their occupational and educational backgrounds: 1) professionals, executives, other highly trained people; 2) clerical and sales workers, self-employed craftsmen, foremen, small businessmen, elementary school teachers; 3) blue-collar workers, service workers, salaried craftsmen. Routine physical exams were performed in the morning, after the subjects had been requested not to smoke or eat after the previous midnight. Audiograms, blood chemistry tests (serum cholesterol and triglycerides, uric acid, glucose tolerance), blood pressure, and heart rates were included in the exams, as well as an assessment of the ear diseases, smoking habits, and noise exposures of the subjects. Subjects with high cardiovascular risk factors had diastolic blood pressures greater than 85 mm Hg, systolic blood pressures greater than 135 mm Hg, serum cholesterol levels higher than 240 mg per 100 ml, and smoked over 10 cigarettes per day. No correlations were found between hearing loss and high cardiovascular risk factors. Group 3, which had both greater hearing loss (sensorineural and conductive) and greater noise exposure did not have increased cardiovascular risk factors.
CARDIOVASCULAR RISK FACTORS AND HEARING LOSS

A Study of 1000 Fifty-Year-Old Men

B. Deetner, H. Hedström, I. Klockhoff and A. Svedberg

From the Department of Audiology, Otorhinolaryngology and Internal Medicine,
University Hospital, Uppsala, Sweden

(Received May 16, 1974)

Abstract. The hypothesis that cardiovascular risk factors might be of importance in the development of senso-
neural hearing loss was tested in a material of 1000 fifty-
year-old men. No significant correlations were found. 
The present study confirmed the well-known observation 
that the left ear is usually poorer than the right. Hearing 
loss in the right ear was found to be related to the 
smoking habits in the group with no history of noise 
exposure. The explanation for this is discussed. Hearing 
loss was more common in social class 3 than in the other 
social classes. This difference was principally referable 
to noise exposure but also to conductive hearing loss. 
A prospective study of this material will further analyze 
the question concerning a possible relationship between 
cardiovascular risk factors and hearing loss.

The deterioration of hearing related to aging is 
a well known phenomenon. It is predominantly 
a matter of progressive sensorineural high tone 
loss, which has been observed to start already 
during the third decade of life. Most of the 
available information on morphological and 
functional changes in presbycusis has been re-
viewed by Schmidt (1967). In summing up he 
points out: "we all carry to our senium the 
cumulation of harms done to our hearing acuity 
in a lifetime."

Among factors discussed are principally noise 
exposure and vascular disease.

The importance of cardiovascular disease is 
unclear. No relation between hearing loss and 
cardiovascular disease was noted by Bunch et 
al. (1929, 1931) or Miller & Oti (1966). On the 
other hand a relationship between a certain 
morphological type of presbycusis and vascular 
disease has been suggested by Schuknacht (1964). 
Weston (1964) reported that the age of one and 
the progress of presbycusis were related to 
circulatory disturbances, among other factors.

Audiological studies performed in different 
areas of the world have indicated a possible 
relationship between hearing loss and high intakes 
of saturated fats, high cholesterol levels, 
esterol cerebrovascular and coronary heart disease (Rosen 

Great attention has focused on Rosen's report, 
according to which a change in the fat composition 
of the diet in the direction from saturated to unsat-
urated fat was followed not only by a redu-
tion of the incidence of coronary heart disease 
but also by a diminished impairment of hearing 
(Rosen et al., 1970b).

An observation which is also of interest in this 
connection is the study by Ismail et al. (1971) 
showing that physical exercise which resulted in 
improvement in different cardiovascular param-
ters, did not affect the hearing thresholds, but 
resulted in an improved ability to recover from 
a temporary threshold shift induced by noise.

Against this background it was judged to be 
of interest to perform tone audiometry in con-
nection with an extensive health examination survey of 
a large unselected material of 50-year-
old men. The health examination was intended 
to identify risk factors for cardiovascular disease, 
such as hypertension, elevated blood lipid 
levels and smoking.

Table I. Factors used to assess hearing loss

<table>
<thead>
<tr>
<th>Factor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td>2.</td>
<td>Diastolic blood pressure</td>
</tr>
<tr>
<td>3.</td>
<td>Heart rate</td>
</tr>
<tr>
<td>4.</td>
<td>Serum cholesterol</td>
</tr>
<tr>
<td>5.</td>
<td>Serum triglycerides</td>
</tr>
<tr>
<td>6.</td>
<td>Uric acid</td>
</tr>
<tr>
<td>7.</td>
<td>Hematocrit</td>
</tr>
<tr>
<td>8.</td>
<td>Glucose tolerance</td>
</tr>
<tr>
<td>9.</td>
<td>Smoking habit</td>
</tr>
<tr>
<td>10.</td>
<td>Noise exposure</td>
</tr>
</tbody>
</table>

The purpose of the study was to assess 
the presence of risk factors and ear 
problems with and without noise 
exposure. It does not seem to have b

MAT

The material consists of fifty, who were con-
sumed health examination survey of 
identifying risk factors 
disease in healthy mid-
Uppsala. The exami-
the morning between the 
participants were asked 
right and not to 
night. The researchers 
whole examination pe-
rate was 83.9. Table I 
which have been used t
ship with the amount of 
loss.

Grouping of the men 
was based on in-
formation. The following 
Andersen et al., 1970):
Social class I: Profes-
sionals, high managers of large busi-
ness entities. Social class II: Cler-
ical, self-employed, proprietors, elementary 
Social class III: Labor 
operators, salaried and
The purpose of the present investigation was to search for possible correlations between hearing loss and cardiovascular risk factors, separate as well as in combinations. Studies concerning smoking habits and hearing function do not seem to have been reported earlier.

MATERIAL

The material consisted of 1,000 men, aged fifty, who were consecutively selected from a health examination survey, with the special aim of identifying risk factors for cardiovascular disease in healthy middle-aged men in the City of Uppsala. The examination was performed in the morning between 7.15 and 9.00 a.m. The participants were asked to come after an overnight fast and not to have smoked after midnight. The researchers were the same during the whole examination period. The participation rate was 83.9 %. Table 1 shows the "risk factors" which have been used to study the interrelationship with the amount of sensorineural hearing loss.

Grouping of the material according to social classes was based on interview report on occupation. The following three classes were used (Andersen et al., 1970):

Social class 1: Professionals and academically trained persons, high officials, proprietors and managers of large businesses and industry.

Social class 2: Clerical and sales workers, foremen, self-employed craftsmen, small business proprietors, elementary school teachers.

Social class 3: Labourers, service workers, operatives, salaried craftsmen.

METHODS

The heart rate was counted and the blood pressure measured after 10 minutes of rest in the lying position.

The serum cholesterol and the triacylglycerines were assayed in an isopropanol extract of serum by using a Technicon dual-channel system (N-24 A and N-70). The hemoglobin was measured with a micromethod with capillary tubes. Uric acid was determined by a wolfzram method.

The intravenous glucose tolerance test (IVGTT) was performed with a glucose dose of 0.5 g per kg bodyweight administered as a 50 % solution. Blood samples for determination of glucose in plasma were taken at 10 min intervals over 1 hour for estimation of glucose tolerance, which was expressed as a K-value calculated from the formula: K = ln 2 x 100/Tᵣ, where the Tᵣ is the time in minutes required for the concentration to be reduced by half its value. This IVGTT was performed in a random sample of 594 men in this study.

The hearing was tested with a pure tone audiometer at the frequencies 500, 1,000, 2,000, 3,000, 4,000 and 6,000 Hz bilaterally. With the sound-insulted headphones the investigation room was found to be silent enough to permit relevant threshold determinations down to 15 dB (ISO, R 389), which was regarded as non-significant hearing loss and lower values were accordingly not registered.

The individuals were interviewed by questionnaire concerning history of ear disease, noise exposure and smoking habits.

CLASSIFICATION OF THE MATERIAL AND STATISTICAL PROCEDURES

A computer IBM 370/155 was used for sorting and calculations. A check was included that all parameters fell within reasonable limits. Correlation studies were performed between hearing loss and the factors in Table 1. Moreover smoking habits, history of noise exposure and of ear disease were included in the statistical calculations.
The material was grouped as follows:

Material A consisting of the total material, 1,000 males.

Material B consisting of 762 males after exclusion from the total material of 238 subjects who had possible conductive hearing loss.

The criteria for this exclusion were: (i) Hearing loss of 30 db or more at 500 Hz in at least one ear, (ii) and/or positive answer to the question “Have you had any ear inflammation after the age of 20”, (iii) and/or positive answer to the question “Have you any ear disease”.

Material B was subdivided according to the following criteria:

Social classes:

1 103 (13.3 %)
2 319 (41.9 %)
3 540 (64.6 %)

History of noise exposure:

BI “yes” 388 (50.9 %)
BII “no” 374 (49.1 %)

The following groups were selected from Material B with regard to smoking habits and noise exposure:

B1. Noise exposure, never smoked, n = 101
B2. No noise exposure, never smoked, n = 105
B3. Noise exposure, heavy smokers, n = 79
B4. No noise exposure, heavy smokers, n = 92

(Heavy smoking means smoking >10 cigarettes per day and smoking duration of at least 10 years. Pipe and cigar smokers were excluded unless they did not smoke >10 cigarettes a day).

Classification of hearing loss

The hearing loss of each individual was classified from the aspects given below. Each classification comprised the computing of hearing loss values of the right ear, the left classification and both ears with respect to the following classification groups 1–7:

1. Sum of hearing loss at 500–6,000 Hz
2. Sum of hearing loss at 1,000–6,000 Hz
3–7. Hearing loss at 1,000, 2,000, 3,000, 4,000 and 6,000 Hz, respectively.

Each group was subdivided into five disease classes of about equal size. Moreover the groups 3–7 were subdivided into three classes concerning each ear:

Hearing loss 15 dB
Hearing loss 20–35 dB
Hearing loss >40 dB

Calculation in material B

The main computing program cross-classified all individuals within material B and its subgroups B1 and BII, i.e., with and without no exposure respectively, regarding each risk factor and all groups of the hearing loss. The $X^2$ test was used to test the significance of differences.

The accepted level of significance was $p < 0.05$.

The individual difference in hearing loss between both ears at 1,000, 2,000, 3,000, 4,000 and 6,000 Hz respectively were calculated for the total group B, subgroup B1 and BII as well as for risk groups B1–B4. The Kolmogorov-Smirnov test was applied to test for any kind of difference in the distribution between B1 and BII and between all combinations of B1–B4.

RESULTS

Coronary risk factors

The correlation studies between hearing loss in material B, classified as presented above, and the factors of Table I showed no significant correlations except for smoking habits which will be discussed below.

No significant correlations were found between the amount of hearing loss in high and low risk groups selected from material B. Subjects in the high risk group (n = 28) had systolic blood pressure 135 mmHg and diastolic blood pressure 85 mmHg, serum cholesterol 240 mg/100 ml and smoked more than 10 cigarettes a day. Subjects in the low risk group (n = 35) were non-smokers, had serum cholesterol 220 mg/100 ml and systolic blood pressure 125 mmHg and diastolic blood pressure 80 mmHg.

Table II shows the distribution of hearing loss in the total material, divided into social classes.

<table>
<thead>
<tr>
<th>Table II. Distribution of hearing loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>of hearing loss in 500–6,000 Hz. dB</td>
</tr>
<tr>
<td>social class</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
</tbody>
</table>

With the screen hearing loss, of hearing loss", we may be regarded in Table II, though well as most peaks which 25.3% had 16.0% were free. The correspond 12.3% and 23.8 in distribution classes 1 and 3, be statistically significant.

It seems that more prevalent class 1. Among sible conducive given previously class 3 and only 11.2% responding the following subjects were 44 and 13.5%, to between the two statistically sign occurrence of m in social class 3.

Noise exposure

In the total material was statistically 3 than in any of...
Table II. Distribution (%) of hearing loss in social classes in 1,000 males, aged 50. The amount of hearing loss is expressed as the sum of dB hearing loss of both ears at five frequencies: 500-6,000 Hz. With the screening level of 15 dB an amount of hearing loss of 150 dB that means no significant hearing loss.

<table>
<thead>
<tr>
<th>Amount of hearing loss in dB</th>
<th>Social 1</th>
<th>150-195</th>
<th>200-245</th>
<th>250-300</th>
<th>300-350</th>
<th>350-400</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>1,000</td>
<td>150</td>
<td>200</td>
<td>250</td>
<td>300</td>
<td>350</td>
</tr>
<tr>
<td>1</td>
<td>130</td>
<td>23.8</td>
<td>24.6</td>
<td>17.7</td>
<td>24.3</td>
<td>12.3</td>
</tr>
<tr>
<td>2</td>
<td>402</td>
<td>33.4</td>
<td>32.0</td>
<td>20.3</td>
<td>15.7</td>
<td>14.7</td>
</tr>
<tr>
<td>3</td>
<td>488</td>
<td>16.0</td>
<td>13.0</td>
<td>21.6</td>
<td>22.2</td>
<td>26.3</td>
</tr>
</tbody>
</table>

With the screening level of 15 dB, a sum of hearing loss of 150 dB means "no significant hearing loss", whereas a sum of 300 dB or more may be regarded as severe hearing loss. As seen in table II, hearing loss was most common as well as most pronounced in social class 3, in which 26.3% had severe hearing loss while only 16.0% were free from significant hearing loss. The corresponding figures in social class I were 12.3% and 23.8% respectively. This difference in distribution of hearing loss between social classes 1 and 3, seen in the table, was found to be statistically significant.

It seems that conductive hearing loss was also more prevalent in social class 3 than in social class 1. Among the 238 subjects, who had possible conductive loss according to the criteria given previously, 53.4% belonged to social class 3 and only 11.7% to social class 1. The corresponding figures among the remaining 762 subjects were 44.6% belonging to social class 3 and 13.5% to social class 1. This difference between the two social classes was however not statistically significant. There was also a greater occurrence of men with a history of ear disease in social class 3 than in the other two classes.

In the total material a history of noise exposure was statistically more prevalent in social class 3 than in any of the other two classes.

In material B there were 388 individuals with a history of civil and or military noise exposure, whereas the remaining 334 cases denied such exposure. The hearing loss was found to be significantly greater in the noise-exposed group, the difference being most pronounced around 4,000 Hz (p < 0.001). These observations were valid for the left as well as for the right ear.

In cases with asymmetrical hearing data (difference > 10 dB) the hearing loss was in most cases greater in the left ear than in the right ear. The number of cases with left-sided inferiority was found to increase towards higher frequencies to be most pronounced at 4,000 Hz. In this respect there was no difference between those who had answered "yes" or "no" to the question concerning noise exposure.

Smoking habits

In the total material there were 509 smokers and 276 who had never smoked. Neither material A nor material B showed any significant differences in hearing loss related to smoking habits per se. However, smoking and hearing loss may still have an etiological common denominator, according to the following.

Combinations of noise exposure and smoking habits

The four sub-groups in material B (B1-B4) listed above were selected for correlation studies between, on one hand, hearing loss and, on the other, history of smoking habits and noise exposure. Hearing data in the four groups are presented in Table III.

Only one indication of a possible effect of smoking was found. Among the 92 individuals (B4) who had smoked more than 10 cigarettes daily but had not been exposed to noise, the amount of right-sided hearing loss was significantly greater (p < 0.001) than in the 105 individuals (B3) who had never smoked and had not been exposed to noise (Figs. 1 and 2). Among these smokers, who were not exposed to noise, there was no right-sided superiority in...
Table III. Hearing loss related to history of noise exposure and smoking habits in 377 males aged 50

<table>
<thead>
<tr>
<th>Hz</th>
<th>Ear</th>
<th>No history of noise exposure</th>
<th>History of noise exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Non-smokers</td>
<td>Smokers</td>
</tr>
<tr>
<td>2000</td>
<td>Right</td>
<td>18.1</td>
<td>19.0</td>
</tr>
<tr>
<td>2000</td>
<td>Left</td>
<td>19.0</td>
<td>19.2</td>
</tr>
<tr>
<td>3000</td>
<td>Right</td>
<td>22.0</td>
<td>23.7</td>
</tr>
<tr>
<td>3000</td>
<td>Left</td>
<td>23.2</td>
<td>23.7</td>
</tr>
<tr>
<td>4000</td>
<td>Right</td>
<td>22.0</td>
<td>23.0</td>
</tr>
<tr>
<td>4000</td>
<td>Left</td>
<td>24.2</td>
<td>24.7</td>
</tr>
<tr>
<td>6000</td>
<td>Right</td>
<td>28.3</td>
<td>29.8</td>
</tr>
<tr>
<td>6000</td>
<td>Left</td>
<td>30.3</td>
<td>31.3</td>
</tr>
</tbody>
</table>

hearing but instead an insignificant tendency towards left-sided superiority. These findings refer to 3000 and 4000 Hz.

No similar differences were found in the noise exposed material between the 79 individuals (H3) who had smoked 10 cigarettes daily and the 101 individuals (H1) who had never smoked.

**DISCUSSION**

The phenomenon that the left ear usually is poorer than the right ear, has been observed previously (Glorig & Roberts, 1965). Asymmetrical hearing impairment of this kind has been ascribed to noise, especially from fire arms. In our study left-sided inferiority, was found not only among the individuals with noise exposure but also in those who denied noise exposure. It must be kept in mind, however, that most of the subjects who denied noise exposure had nevertheless had a period of military service in their past. The left-sided inferiority, statistically observed, might also be a more genuine origin since the phenomenon has been observed among male children as well (Kannan & Lipscomb, 1974).

Another observation, partly connected to noise exposure, was that hearing impairment was more common in social class 3 than in other classes. This difference was found to be due not only to noise exposure but also to greater occurrence of conductive hearing loss.

A correlation between hearing impairment and cardiovascular risk factors has been discussed, not least considering the publications of Rosen et al. (1965, 1970). They reported that a long-term change of the fat diet from saturated to polyunsaturated fat intake was followed by a decrease not only in the incidence of coronary heart disease but also in the deterioration in hearing. In our study we did not find any correlation between hearing loss and cardiovascular risk factors or combinations of risk factors.
the only exception was the peculiar finding that the hearing in the right ear was poorer in men who had never smoked than in the individuals who had never smoked. This difference was statistically significant. Among the individuals with a history of noise exposure no similar difference was found between the heavy smokers and those who had never smoked. We cannot find any obvious explanation for this observation. It may be possible that smoking, or associated conditions in heavy smokers, can result in a deterioration of hearing, with an effect only demonstrable in the absence of noise trauma. Why such a hypothetic effect should involve predominantly the right ear, which otherwise usually prefers exposure, is not necessarily a matter of causality. The question of a possible relationship between cardiovascular risk factors and hearing exposure is thus motivated further investigation, especially in a prospective character. The follow-up of our study service in a statistically well-defined group of men will offer such a possibility.

ZUSAMMENFASSUNG

Die Hypothese, dass kardiovaskuläre Risikofaktoren für die Entwicklung der Schwerhörigkeit von Bedeutung sein könnten, wurde an 1000 30-jährigen Männern getestet. Keine signifikanten Korrelationen wurden festgelegt. Die vorliegende Studie stützt die bereits bekannten Befunde, dass das linke Ohr gewöhnlich mehr betroffen ist als das rechte, was wahrscheinlich durch die Inwirkung einer Hormonenspiegelung in den Gruppen mit Lärmschäden in der Allgemeinheit belegt wurde. Die Ergebnisse der Studie könnten unter anderem helfen, Präventionsmaßnahmen in der Gruppe der 30-jährigen Männer aufzudecken. Es wird betont, dass eine Verbindung zwischen kardiovaskulären Risikofaktoren und Gehörschäden vermutet werden kann.

REFERENCES


I. Klukkoff, M.D.
Dept. of Otolaryngology
University Hospital
Uppsala 14 Sweden
## SUMMARY FORM FOR

**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>G.A. Froehlich, Col. GAF, NC</td>
<td>German Air Force Institute of Aviation Medicine Fuerstenfeldbruck</td>
</tr>
</tbody>
</table>

<table>
<thead>
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<th>Investigator's Phone No.</th>
<th>Sponsoring Organization</th>
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<tbody>
<tr>
<td>Same as above</td>
<td>Same as above</td>
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</table>

### Citation

<table>
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<th>θ of Ref. No.</th>
<th>θ of Fig. No.</th>
<th>Language</th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>7</td>
<td>English</td>
</tr>
</tbody>
</table>

### Type & duration of experiment
Laboratory experiment—short term

### Purpose for study
To test 3 different ear protectors used by the German AF, and determine their effectiveness in reducing autonomic responses produced by aircraft and impulsive noise.

### Description of test groups (subjects, θ etc.)
1 group of 25 human subjects with normal hearing; served as their own controls.

### Control of other stresses
Subjects were blindfolded, and possible random noises were masked by a continuous 50 db(A) white noise.

<table>
<thead>
<tr>
<th>Noise Stimulus</th>
<th>Statistical Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source: child's pistol (impulse noise)</td>
<td>CVS Response Measured</td>
</tr>
<tr>
<td>Jet aircraft noise</td>
<td>(1) peripheral blood flow with digital plethysmography finger pulse amplitude (2) heart rate</td>
</tr>
<tr>
<td>Spectral characteristics: see graph</td>
<td>Nonauditory effects (no ear protectors):</td>
</tr>
<tr>
<td>Centered at 1000 Hz</td>
<td>(1) Pulse Amplitudes—20 sec background amplitude decreased to 63.3% initially; back to 84.4% at end of 20 sec.</td>
</tr>
<tr>
<td>Noise level: impulse—130 db; 95 db (aircraft)</td>
<td>(2) Heart rate—no significant changes due to noise.</td>
</tr>
<tr>
<td>Length of exposure: 20 sec (aircraft)</td>
<td>Other: electrodermal responses—appearance of voltage due to noise.</td>
</tr>
</tbody>
</table>

### Number of trials
Not specified

### Author's conclusions
The best ear protectors (with which the amplitude response was lessened) were those that blocked out the low and medium frequencies. All 3 blocked out high frequencies well. It was concluded that autonomic nervous system responses are more affected by low and medium noise frequencies.

### Evaluation & comments
This is one of the many studies that shows peripheral vasoconstriction as a response to noise.

The effectiveness of 3 different ear protectors in blocking autonomic responses to noise was studied in a group of 25 human subjects with normal hearing. The responses measured were heart rate, fingerpulse amplitude, and electrodermal response. Each subject was blindfolded and a background of continuous 50 dBA white noise was present throughout the study to eliminate other sensory stimuli. The subjects served as their own controls—responses with and without ear protectors were recorded. The noise doses were of 2 types: impulse noise from a child's pistol at a level of 130 dBA and 20 sec. of jet aircraft noise at a level of 95 dBA with spectra centered at 1000 Hz. No significant changes were noted in heart rate due to noise. Definite electrodermal and fingerpulse amplitude responses due to noise were observed. With no ear protectors, there were fingerpulse amplitude reductions of 63.3% plus or minus 12.6% from the jet aircraft noise and 62% plus or minus 14% from the impulse noise. The ear protectors that were most effective in reducing the fingerpulse amplitudes and the electrodermal responses to noise were also more efficient in blocking the low and medium noise frequencies. Since all 3 ear protectors could block high frequencies equally well, it was concluded that the autonomic nervous system responds more to low and medium noise frequencies.
THE EFFECTS OF EAR PROTECTORS ON HUMAN PHYSIOLOGICAL RESPONSES TO ACOUSTIC- AND SIMULATED NOISES

O. E. FREIDIG, Gen., USAF,

FIRESTONE, INVESTIGATOR

INTRODUCTION

After extensive studies of the aural effects of noise, in recent years there has been an increasing interest in the non-auditory physiological effects of noise on man. The first investigators were primarily concerned with the effects of intense noise on the circulatory system. Here we encounter the most reliable finding peripheral vasomotorisation together with more variable other cardiovascular changes. In conjunction with respiratory and endocrinological changes, these effects are physiological responses within the frame of aeroacoustic mechanisms.

Since all these responses depend primarily on the intensity, duration and spectral character of noise, the wearing of ear protectors must decrease the physiological responses. Therefore we have chosen an acoustical stimulus simulating noise and jet aircraft noise together with three different types of ear protectors currently in use in the German Armed Forces (Fig. 1).
As typical aircraft noise we have used a 20 sec P-106(3) jet aircraft noise of 95 dB(A) +
95 dB(A), as demonstrated in Fig. 3. Since there is a discrete frequency in the octave band
centered at 1000 Hz, there is a very annoying, shrilling sound within a broadband noise.

The noise levels and spectra in the subjects' ear canals were calculated by subtracting from
the ambient noise level the attenuation values of the three ear protectors.

The peripheral blood flow was determined by use of photoelectric transducers at the metacarpus of
the right middle finger. From these plethysmographic traces we could also compute the pulse
frequency (Fig. 4).
The electrodermal response (EDR) is the biphasic appearance of a voltage in response to an emotional stimulus. It reflects already minor changes in activity of the autonomic nervous system. The total plane of positive and negative phases were computed in mV. All tests have been conducted in a room having normal reverberation at 22 ± 5°C and noise between 18 - 1600 hours. In order to eliminate other sensory stimuli, the subjects had been blindfolded and possible random noise had been masked by a continuous 50 dB(A) white noise.

RESULTS

1. The Effects of Ear Protectors on Peripheral Vasopressinase Cased by a 25 sec Jet Noise (Fig.5)

Taking the average of the last 10 amplitudes before the sudden onset of noise an 100%,
without ear protectors we have an amplitude reduction to 63.3% ± 11.5% as initial response and towards the end of the stimulation a recovery to 84.4% ± 13%. The amplitude reductions are considerably lower when ear protectors are used. With ear protectors the amplitudes are reduced to 75% ± 12% and recovery to 99%. The responses with the ear protectors are essentially the same: initial response 75% ± 12% and 99% ± 9% towards the end. The ear protectors offer less protection as it is expressed by the mere marked amplitude reductions to 72% ± 12% as initial response and a recovery to 90% ± 9% at the end of the 20 sec period. The differences of responses with and without the various ear protectors have been significant at the 0.001 level.

![Fig. 5](image.png)

Peripheral vasopressinase caused by 95 dB(A) jet noise.
(a) pre-exposure = 100% (b) initial response and (c) recovery to the end of exposure.

2. Effects of Ear Protectors on Peripheral Vasopressinase Cased by Impulsive Noise (Fig.6)

The initial responses after the 150 dB peak static without ear protectors show marked amplitude reductions to 62% ± 14%, with the use of ear protectors there is only a small reduction to 68% ± 3% and with ASPIRATION X to 72% ± 20%. In every interindividual comparison, the protective

![Fig. 6](image.png)

Peripheral vasopressinase caused by 113 dB impulsive noise.
(a) pre-exposure = 100% (b) without ear protector, (c) pre-exposure and (d) ear protectors
3. The Effects of Ear Protectors on the Electrodermal Response (EDR) (Fig. 2)

As already mentioned by several authors, there are few significant changes of the heart rate under the stress of noise, as can be seen in the following table:

**Table I:** Changes of Heart Rate in % with 95 dB(a) Noise

<table>
<thead>
<tr>
<th>Condition</th>
<th>CON-PIE</th>
<th>ELLISON</th>
<th>SELECTOR K</th>
<th>WITHOUT</th>
</tr>
</thead>
<tbody>
<tr>
<td>CON-PIE</td>
<td>2.1%</td>
<td>1.1%</td>
<td>1.8%</td>
<td>1.5%</td>
</tr>
<tr>
<td>ELLISON</td>
<td>2.5%</td>
<td>1.5%</td>
<td>1.8%</td>
<td>1.7%</td>
</tr>
<tr>
<td>SELECTOR K</td>
<td>2.6%</td>
<td>2.1%</td>
<td>1.9%</td>
<td>1.6%</td>
</tr>
<tr>
<td>WITHOUT</td>
<td>2.9%</td>
<td>2.2%</td>
<td>2.1%</td>
<td>1.9%</td>
</tr>
</tbody>
</table>

**Table II:** Changes of Heart Rate with 130 dB Impulsive Noise

<table>
<thead>
<tr>
<th>Condition</th>
<th>CON-PIE</th>
<th>ELLISON</th>
<th>SELECTOR K</th>
<th>WITHOUT</th>
</tr>
</thead>
<tbody>
<tr>
<td>CON-PIE</td>
<td>0.1%</td>
<td>1.5%</td>
<td>1.5%</td>
<td>1.0%</td>
</tr>
<tr>
<td>ELLISON</td>
<td>0.4%</td>
<td>1.5%</td>
<td>1.6%</td>
<td>1.2%</td>
</tr>
<tr>
<td>SELECTOR K</td>
<td>0.5%</td>
<td>1.7%</td>
<td>1.5%</td>
<td>1.3%</td>
</tr>
<tr>
<td>WITHOUT</td>
<td>0.6%</td>
<td>1.8%</td>
<td>1.4%</td>
<td>1.5%</td>
</tr>
</tbody>
</table>

4. Subjective Assessment of Ear Protectors Against F-104 Noise

Immediately after the tests, each of the 25 subjects was asked to assess the effectiveness of the three different ear protectors and to establish an order of rank:

<table>
<thead>
<tr>
<th>ORDER OF RANK</th>
<th>SELECTOR K</th>
<th>CON-PIE</th>
<th>ELLISON</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>2.</td>
<td>2</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>3.</td>
<td>1</td>
<td>4</td>
<td>2</td>
</tr>
</tbody>
</table>

The CON-PIE ear plug has been assessed as the most effective ear protector, closely followed by the ELLISON earplug. In accordance with the results of objective measurements, the SELECTOR K earplug is assessed as considerably less effective.
DISCUSSION

As shown by the frequency analysis in Fig. 3, marked autonomic response had to be expected. They were studied for the peripheral blood flow as well as the electrodermal responses. Contrary to this, the heart rate showed no significant change in terms of increase or decrease. The autonomic characteristic of the three ear protectors are quite different, so that at the median of the subjects the intensities and quality of the noise had been different too. By calculation, the protective effects of the SILOCON earplug is best because the predominant frequencies above 1000 Hz are attenuated most effectively. The second best is the CON-SIL earplug whose attenuation at 1000 Hz is not as effective, but better in the lower frequency range. SILOCON E has the least protective effect of all three in the lower and medium frequency range, whereas above 1000 Hz the attenuation properties are close for all three ear protectors. These differences in autonomic response must be due to the different noise levels in the lower and medium frequency range.

While the exposure to F-104 noise without ear protectors is within the area of potential acute effects (Fig. 5), the proper use of the ear protectors reduces the exposure to the area of only possible autonomic responses. All amplitudes reductions as compared to pre-exposure amplitudes are significant at the 0.001 level as well as the differences without-with ear protectors. The protective effects of CON-SIL and SILOCON are essentially the same and both are significantly better than those of SILOCON E. More important than the short-term initial effects are those towards the end of the 20 ear noise exposure period, since they indicate the consistent response level for noise exposure of longer duration. Here as well, it is noted, that with CON-SIL and SILOCON the responses return to pre-exposure values during noise exposure, whereas with SILOCON E a small vasoconstriction will still remain. The short-term initial response may be due to a certain startling effect at the rapid onset of noises. This might be supported by the appearance of marked electrodermal response without ear protectors and a considerable decrease of this response by the use of SILOCON, CON-SIL and finally SILOCON E. The standard deviations for the ECG are much larger, which makes the measurement of finger pulse amplitudes a more reliable parameter.

There was no fixed intradividual relationship between the two test parameters inasmuch as under identical conditions strong responses in one parameter did not necessarily mean strong responses in the other one.

ACKNOWLEDGEMENT

Thanks are due to Dr. VITE for compilation and evaluation of statistical data and to Mr. KLEIBER for his assistance and guidance in the preparation of the English version. Both are assigned to our Institute.
SECTION 7
## Summary Form for Studies on the Effects of Noise on the Cardiovascular System (CVS)

### Principal Investigator(s):
Prof. Sanford E. Gerber, Anthony Nulce and N. Elias Lamb

### Institution and address where research was performed:
Department of Speech, University of California, Santa Barbara, Calif. 93106

### Investigator's Phone No.: University of California

### Sponsoring Organization:

### Citation:

### # of Ref.'s: 18

### # of Fig.'s: 5

### Language: English

#### Purpose for Study:
To test the validity of the cardiovascular response to noise and to see if this response varies with the sound pressure level.

#### Type & duration of experiment:
Laboratory experiment in sound-shielded room, short-term.

#### Description of test group [subjects, n, sex, age, etc.]:
Group: 14 women, 21-32 years of age, all not menstruating at time of experiment. No separate control group. Subjects tested during chair-rest and requested to make no overt responses to the noise stimuli.

#### Control of other stressors:
Laboratory conditions used - no other source.

#### Noise Stimulus:
- Source: introduced through earphones.
- Spectral characteristics: Narrow band noise centered at 1500 Hz (graph included).
- Noise level: 20, 40, 60, 80 dB in short bursts (signals).
- Length of exposure: 1 second per signal.
- # of trials: Each trial: 4 bursts of noise (1 sec. each) l interval of quiet at a random point in the trial with 30 sec. in between.

#### Author's conclusion:
The heart rate response is independent of the sound pressure level and is nonphasic-deceleration than audiometric method.

#### Evaluation & comments:
A separate control group would have made the results more meaningful, since normal heart rate changes during quiet could have been compared to those during exposure to short bursts of noise.

The use of a cardiovascular response (heart rate change) to noise as an audiometric method was evaluated in 14 nonmenstruating women with normal hearing, aged 21-23 years. The pre-stimulus heart rates of the subjects were compared to the post-stimulus rates, such that the subjects served as their own controls. The subjects were tested while seated quietly in a chair in a sound-shielded room. The noise stimuli consisted of bursts of narrow-band noise (signals) centered at 1000 Hz and dropping 22 dB/octave (a graph is included). Noise signals of 20, 40, 60, and 80 dB, lasting for 1 second each, were introduced through earphones. Each trial consisted of 4 one second bursts of noise (signals) at a given sound level interspersed randomly with 1-second intervals of quiet periods (non-signal), lasting for the same total time as the noise signals. The 4 bursts and 4 quiet periods were presented in 3 different arrangements at each sound pressure level consecutively, beginning with the 20 dB as follows:

1) 4 bursts 20 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules;
2) 4 bursts 40 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules;
3) 4 bursts 60 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules;
4) 4 bursts 80 dB (1 sec each) + 4 quiet periods (1 sec each) in 5 presentation schedules.

The finger tip pulse volume was continuously monitored during the tests. The heart rates averaged over 3 pulse cycles were measured with a Hewlett-Packard heart rate finder for all signal and non-signal events. A three-way analysis of covariance with the pre-stimulus heart rate as the covariant was used to analyze the data. Significant heart rate differences were found between the signal (noise) and non-signal (quiet) conditions. The differences in heart rates were independent of the noise level (an all-or-none response). The heart rate response to the noise signals was monophasic—an early deceleration after the noise burst and a gradual return to the pre-stimulus rate. The pre-stimulus rates were from 53 to 110 beats/minute with a mean of 79.6 beats/minute and a standard deviation of 11.2 beats/minute. The mean decrease in heart rate following the noise bursts was 1.46 beats. Significant differences in the heart rate responses were also found between subjects due to noise. The authors conclude that the cardiovascular response to noise is a valid audiometric method, although significant individual differences in responses are encountered.
The Cardiovascular Response to Acoustic Stimuli

SANFORD E. GERBER, ANTHONY MULAC and M. ELISA LAMBI

University of California, Santa Barbara, Calif.

Key Words. Cardiovascular · Heart rate response · Autonomic response

Abstract. The purposes of this investigation were to determine whether adults display alterations of cardiac rate under acoustical stimulus conditions and whether such alterations are influenced by signal level. The stimulus consisted of a narrow band of noise centered at 1,000 Hz and presented at 20, 40, 60 and 80 dB SPL. The stimulus was found to produce alterations of heart rate significantly different from variation under non-stimulus conditions, indicating that cardiovascular responses occurred. However, the responses themselves were unaffected by differences of sound pressure level.

Electrophysiological audiometry employs responses to acoustic stimulation manifested by observable changes of some physiological property of the subject, while behavioral audiometry requires an overt bodily reaction. One would choose electrophysiological procedures for the difficult-to-test patient: an infant, a multiply handicapped person, a severely retarded person. Of the electrophysiological procedures, cardiovascular response audiometry may be preferable because (unlike some others) it does not require a noxious stimulus, nor does it necessarily assume an intact central nervous system. However, the properties of the cardiovascular response are not well understood; and, until they are, its utility as an audiological method must be viewed with caution.

Attempts have been made to assess the cardiovascular response as a means of measuring hearing sensitivity, although heart rate has been only one of

Audiology 15: 1–10 (1977)

An abbreviated version of this paper was presented at the annual convention of the American Speech and Hearing Association meeting in Las Vegas, Nev., November 1974.
several autonomic responses investigated. Unfortunately, the heart rate response itself has been defined in a variety of ways, leading to discrepancies in the reported forms of the response. A definition is needed which accounts for the differences in cardiovascular behavior between stimulus and non-stimulus conditions since, in order for a change of cardiovascular rate to be considered a response to an environmental stimulus (rather than the normal variation of heart rate), it must be significantly different from those changes which occur in the absence of external stimulation. The difference between stimulus and non-stimulus conditions may be assessed by comparing heart rate changes under these two conditions when other variables are controlled. However, in investigations of the effects of signal intensity, this non-stimulus condition has rarely been included (e.g., Davis et al., 1955; Uno and Grings, 1965) with the notable exception of ZEAMAN and WAGNER (1956) who claimed that they could distinguish responses to suprathreshold signals from responses to signals ‘48 dB below threshold’ [sic]. The failure to include a non-stimulus condition makes it difficult to assess the discrepancy between Uno and Grings’ [1965] finding that the magnitude and latency of the response varied with signal level and Smith and Strawn-Rudge’s [1968] finding that no significant differences occurred for two signal levels 40 dB apart.

Similarly, disagreement about the nature of the heart rate response and its measurement has resulted in different findings regarding the form of the response. Davis et al. [1955] measured pressure pulse, volume pulse, and interpulse interval as responses to acoustic and tactile stimuli, and found a biphasic response consisting of an initial decrease of interval (i.e., increase of rate) with an accompanying decrease of pressure, followed by increases of pressure and interval which exceeded the pre-stimulus values. However, Hogan [1970] found a monophasic response having the form of deceleration of rate followed by recovery to pre-stimulus level, but not beyond. Such discrepancies may have been due to a failure to clearly state what stimuli were employed, what response intervals were used, what measurements were made, and what controls were employed.

Finally, the validity of much of this research is questionable in light of Lacity’s [1966] work on quantifying the heart rate response through studying the influence of the pre-stimulus rate upon ensuing cardiovascular behavior. He adjusted for the law of initial values [Wilder, 1950] through regression

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4 The monophasic form of response is defined as a deviation in either direction without returning to or beyond the pre-stimulus rate; a biphasic response varies above and below the pre-stimulus value.
The Cardiovascular Response to Acoustic Stimuli

analysis, arguing that, since the response is dependent upon the pre-stimulus rate, comparison cannot properly be made among different response events without taking into account the pre-stimulus rate. It follows that a similar procedure is essential for the evaluation of response validity.

Clearly, controversy exists regarding the parameters of the heart rate response of adults to auditory stimulation. Studies which have incorporated control conditions of some type have seemed to indicate that the heart does respond to environmental acoustic events, but that the properties of this response as reported are as varied as the research designs which have purported to measure them. Especially confusing have been the results of the effects of signal intensity. The purposes of the present investigation were to assess the validity of the acoustic cardiovascular response and to determine whether that response is affected by sound pressure level.

Method

In an effort to control for the confounding variables discussed above, the following steps were taken: (1) changes of heart rate following signals were compared with heart rate changes following randomly placed periods of no stimulation; (2) subjects were included as a factor in the experimental design to assess individual differences of pattern of heart rate change; (3) to control for the law of initial values, pre-stimulus heart rate was used as a covariate to analyze post-stimulus heart rate change.

Subjects. 14 females between the ages of 21 and 23 years (mean = 22.4) served as subjects. All reported negative otological histories and passed a 55 dB HTL [ANSI, 1969] screening test on the experimental stimulus. Because of the possibility that physiological changes accompanying menstruation affect hearing [Glass, 1972], all subjects were scheduled at other times of the month.

Stimu. Signals selected as test stimuli were bursts of narrow-band noise centered at 1,000 Hz and dropping 22 dB/octave (fig. 1). These were presented diotically through earphones with each burst having a stimulus rise time of 0.5 ms and a duration of 1 s. 4 signal levels were used: 20, 40, 60, and 80 dB above 20 µN/m² (20 µPa). A 5th test condition was a 1-second interval in which no signal was presented. Hence, the stimulus conditions were 4 bursts of noise and 'blank', arranged in 2 different presentation schedules in which the blank condition occurred as frequently as the combined signal conditions. The non-signal events were distributed randomly among the signal stimuli which occurred in a prescribed order; each condition was presented 5 consecutive times in order of increasing intensity. The test was begun with 5 presentations of 20 dB bursts, with blank events randomly intermixed. Furthermore, the random insertions of the non-signal events were different for each of the 2 presentation schedules assigned to subjects; all schedules except 1 were used 3 times.
*Recording.* Testing was conducted in a darkened, sound-shielded room (General Acoustics Corp.). Subjects were seated with one wrist taped to a table. The finger tip pulse volume transducer (Glascon FP-6) and earphones (Telephonic TDH-49) were placed. Subjects were instructed to avoid overt responses to the signals and to refrain from unnecessary movement. During all phases of the testing, subjects remained awake.

The output from the pulse volume transducer was recorded on a polygraph (Glascon MI) at a writing speed of 25 mm/s. An event marker recorded signal and non-signal presentations on the polygraph paper (Fig. 2).

*Measurement.* Heart rate measurement was done in the manner suggested by Lewis [1971] and used successfully in our laboratory (Golstein, 1972; Grant, 1973; Grant, et al., in press); the onset of each stimulus event marked the point from which the pra-
The Cardiovascular Response to Acoustic Stimuli

stimulus and post-stimulus heart rate measures were taken. A Hewlett-Packard heart rate finder was employed to measure the heart rate averaged over 3 pulse cycles. (It should be noted that this method of analysis could obscure any biphasic response occurring during the first post-stimulus interval.) As figure 2 demonstrates, the pre-stimulus rate was determined by measuring backward from the pulse peak nearest signal onset. This defined the interval called \( P_0 \). The 3 post-stimulus heart rates were determined in the same way measuring in beats per minute forward from signal onset, and were called \( P_1 \)–\( P_3 \). These measures were made for every signal and non-signal event, with events separated by thirty seconds. Heart rates were then converted into change scores from pre-stimulus rate to each post-stimulus rate. In other words, the heart rate changes between \( P_0 \) and \( P_1 \), \( P_2 \), and \( P_3 \) ... \( P_n \) and \( P_{n+1} \) were computed for analysis.

Data analysis. Two separate data analyses were employed, each utilizing a three-way analysis of covariance with repeated measures (Winer, 1962) in which the covariate was pre-stimulus heart rate (\( P_0 \)) before each signal or non-signal event. The efficacy of regression analysis to control for the law of initial values (Winer, 1955) was suggested by Lucey (1956). This statistical procedure measures the relationship between post-stimulus rates from pre-stimulus rates. A practical application of this principle of controlling for the initial value in analysis of covariance. This permits the adjustment of post-stimulus rates to the extent they are related to pre-stimulus rates, and has been employed for this purpose in previous studies (Benjamin, 1963; Graeme et al., in press).

Changes from \( P_0 \) to other time intervals (\( P_1 \), \( P_2 \), ..., \( P_n \)) were represented as signed change scores in beats per minute. Subjects and post-stimulus measurement intervals were also evaluated as potential sources of variation. For each of the two covariance analyses, there were 14 subjects and 5 time intervals. In the first analysis, possible differences among the 4 intensity levels were investigated; in the second analysis, the 4 signal levels combined were compared to the non-signal events in order to assess the validity of the cardiovascular response to acoustic stimuli.

Results

Subject pre-stimulus heart rates ranged from 53 to 110 beats/min, with a mean rate of 79.6 and a standard deviation of 11.2 beats/min. These initial heart rates for each test event served both as \( P_0 \) for computation of change scores that event and as the covariate score for that event.

Effect of Intensity

Results of the three-way analysis of covariance (14 subjects × 4 intensities × 5 time intervals) on data from the signals of 4 intensities (table 1) indicated the following: (1) signal intensity was not found to be a significant source of variance; (2) the subjects themselves were found to be a source of variance (\( p < 0.001 \)); (3) post-stimulus change across time intervals (response latency) was also seen to be a source of variation (\( p < 0.001 \)); (4) no interactions were
found among the 3 independent variables. The lack of subject × time interval interaction indicated that the post-stimulus pattern of change did not differ among individuals over time; the lack of intensity × time interactions showed that the time of maximum heart rate change was unaffected by signal level.

The possibility that rapid habituation within each stimulus level might have masked a differential response to the 4 intensity levels was assessed through a separate two-way analysis of covariance with repeated measures. Heart rates following the first presentation of each intensity level were the data for this 4 (intensities) × 5 (time intervals) analysis. Results failed to show significant differences in heart rate changes for the 4 intensities (p > 0.10).

**Response Validity**

Since the first analysis of covariance revealed no difference as a function of signal intensity across signal events, these data were grouped for comparison with the non-signal events. In this second analysis of covariance (14 subjects × 2 signals × 5 time intervals), a statistically significant difference

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**Table 1. Results of three-way analysis of covariance of heart rate change scores for 4 signal intensity levels**

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>SS</th>
<th>d.f.</th>
<th>MS</th>
<th>F</th>
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<tbody>
<tr>
<td><strong>Between subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A (subjects)</td>
<td>7,305.31</td>
<td>13</td>
<td>561.55</td>
<td>9.65*</td>
</tr>
<tr>
<td>Subj. w. groups</td>
<td>3,202.48</td>
<td>35</td>
<td>88.23</td>
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<tr>
<td><strong>Within subjects</strong></td>
<td></td>
<td></td>
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<tr>
<td>B (Intensity)</td>
<td>190.90</td>
<td>3</td>
<td>63.63</td>
<td>1.01</td>
</tr>
<tr>
<td>AB</td>
<td>4,518.62</td>
<td>79</td>
<td>113.85</td>
<td>1.88</td>
</tr>
<tr>
<td>B × subj. w. groups</td>
<td>10,166.54</td>
<td>145</td>
<td>67.46</td>
<td></td>
</tr>
<tr>
<td>C (time interval)</td>
<td>406.96</td>
<td>4</td>
<td>101.74</td>
<td>5.48*</td>
</tr>
<tr>
<td>AC</td>
<td>1,221.01</td>
<td>52</td>
<td>23.84</td>
<td>1.26</td>
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<tr>
<td>C × subj. w. groups</td>
<td>4,683.43</td>
<td>220</td>
<td>18.55</td>
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<tr>
<td>BC</td>
<td>356.07</td>
<td>12</td>
<td>80.50</td>
<td>1.59</td>
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<tr>
<td>ABC</td>
<td>2,609.54</td>
<td>132</td>
<td>17.15</td>
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<tr>
<td>BC × subj. w. groups</td>
<td>12,487.01</td>
<td>660</td>
<td>18.92</td>
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</tbody>
</table>

* p<0.001.
1 Pre-signal rate was used as the covariate.
between these two signal conditions was taken as an indication of heart rate response to acoustic stimuli as distinguished from normal variation.

Results of this analysis (table II) indicated the following: (1) a significant difference was found between signal and non-signal events \((p<0.001)\). The signal response was monophasic, with deceleration followed by return toward the pre-signal rate (fig. 3); (2) subjects were again found to be a significant source of variance \((p<0.001)\); (3) time interval was again a significant source of variation \((p<0.001)\); (4) no interactions were found among the 3 independent variables.

Discussion

We set out to investigate the validity of the cardiovascular response to sound in adults and to assess the influence of signal level upon it. This study differed from previous research in that the validity of the response was itself a major issue. The data indicated that cardiovascular rate changes constitute a valid response to acoustic stimuli of the type tested here. Further, the response to the stimuli appears to be of the 'all-or-none' type since it was not differentially affected by signal level. Also, we observe that the response is monophasic, beginning with early deceleration after stimulus onset followed by gradual recovery toward the pre-stimulus rate. We pointed out earlier that the analysis of heart beats in groups of 3 could obscure a biphasic response occurring within the first post-stimulus interval. In light of the magnitude of the decelerative response observed during that time interval \((\text{Mean} = -1.46\) beats\()\), it appears unlikely that an acceleration of sufficient size occurred during the first post-stimulus beat to make this hypothesis tenable.

These conclusions support the earlier results of Smith and Strawbridge [1968] who found no differences of cardiovascular rate variation as a function of signal level. They employed a range of 40 dB, while we used a 60 dB range; however, in neither case was the customarily defined threshold of hearing assessed. While we have found that the cardiovascular response to sound is of the 'all-or-none' type, we do not yet know how close to threshold it may be elicited, but it must be less than 20 dB. In an earlier study at our center, Appelbaum [1971] was unable to evoke cardiovascular responses from adults at the previously determined audiometric threshold. Therefore, we conclude that the response may be evoked somewhere near to, but above, threshold; and that it is not altered by further increases of sensation level.
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through 80 dB. Also, our results indicate that the form of the response at
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that
the response of a subject past the age of early Infancy takes a monophesic
form of deceleration followed by return toward the pm-stimulns rate. However, this finding is contrary to the observations of D^v_ et aL [1955Jthat
accelerationoccursfirst.
Ourstatistical analyses of the deta lead us to believe that the test method
caution in its clinical application. While we may have determined the form
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early during the first year of life [GOLDSTEIN,
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importantand conucptually
interesting.
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adequacy, andareencouraged
by ourfindings,
we remain cautious as to their implicationS.

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venant en dehors des stimulations sonores; ces résultats montrent l'existence de réponses cardio-vasculaires. Cependant, les réponses n'étaient pas affectées par les différences de niveau de pression acoustique.

References


Prof. SANTROINTER E. GERBER, Department of Speech, University of California, Santa Barbara, CA 93106 (USA)
### Summary Card

**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th><strong>Principal Investigator(s)</strong></th>
<th>Barbara Griesahn (Dr. Med.)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Institution and address where research was performed</strong></td>
<td>Arbeitsgruppe Exp. Arbeitsmedizin, Institut fuer Hygiene und Arbeitsmedizin, Universitaetsklinikum der Gesamthochschule Essen; D-4500 Essen Nufeldstrasse, 55 Federal Republic of Germany</td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th><strong>No. of Ref.</strong></th>
<th>17</th>
<th><strong>No. of Fig.</strong></th>
<th>5</th>
</tr>
</thead>
</table>

**Purpose for study:** To test fingerpulse amplitude responses to sonic booms with respect to boom intensity, sleep stage, preboom pulse rate, temp., length of experiment, length of quiet time before boom.

**Description of test groups (subjects, n)**
- 2 persons for Part 1
- 2 persons for Part 2

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**Control of other stressors:** Laboratory conditions used temp. varied 170 G - 23.50 C

**Noise Stimulus:**
- Source: sonic boom—reproduced through sonic boom channel into test chamber
- Spectral characteristics: not given
- Noise level: 83.5 dBA average
- Length of exposure: 300 sec. per boom between 10:30 PM and 1 AM.
- N of trials: 2, 4, 8, or 16 booms per night.

**Authors' conclusions:** The decrease in fingerpulse amplitude was not correlated with sonic boom intensity as is generally observed, probably due to the short rise time of the boom. The only variable for which a significant relationship was found was the preboom pulse rate. As the law of initial values states, the greater the preboom pulse rate, the lesser the change in pulse rate due to noise.

**Evaluation & comments:** The small number of subjects is a weak point. No description (age, sex, health) of test subjects is given.
Griffahm, B. Effects of sonic booms on fingerpulase amplitudes during

The fingerpulase amplitudes during sleep of 4 human subjects were studied
after sonic booms of varying intensities between 0.48-1.45 mbar (1 mbar
or 83.5 dBA on the average). The experiments were conducted while the
subjects slept in a test chamber between 10:30 p.m. and 3 a.m., during
which the sonic booms were introduced through an opening in the chamber.
The effects of the following variables on the fingerpulase amplitude
response to noise were studied: preboom pulse rate, sleep stage (monitored
by EEG), interval between booms, duration of experiment, and ambient
temperature (varied from 17°C to 23.5°C). The experiment was divided
into two parts with two subjects in each. Part 1 consisted of 19 nights
as follows: 3 nights quiet; 11 nights with 2 or 4 sonic booms; 6 nights
quiet. Part 2 consisted of 23 nights as follows: 3 nights quiet; 30
nights with 2 or 4 sonic booms; 10 nights quiet; 4 nights with 8 or 16
sonic booms; 6 nights quiet. The interval between booms varied with the
number of booms: 2 booms - 40 min. interval; 4 booms - 20 min.; 8 booms -
3.6 min.; 16 booms - 4.6 min. Each sonic boom lasted for 300 msec. The
fingerpulase amplitudes decreased within 3 sec. after the onset of the
sonic boom, reached a minimum at 8 sec., and then increased toward the
preboom value. The intensity of the sonic booms had no effect on the
degree of fingerpulase amplitude response, which is contrary to previous
experiments in which increased intensity of noise is correlated with a
greater decrease in amplitude. The rise time of the sonic boom may have
been too short to allow for this response. The only variable studied
that affected the fingerpulase amplitude responses to the sonic booms was
the preboom pulse rate. The greater the preboom pulse rate was, the
smaller the decrease in amplitude (the law of initial values).
Effects of Sonic Booms on Fingerpulse Amplitudes during Sleep

BARBARA GRIEFSAHN
Arbeitsgruppe "Experimentelle Arbeitsmedizin" (Leiter: Prof. Dr. med. Dr. phil. Gerd Jansen) im Institut für Hygiene und Arbeitsmedizin (Direktor: Prof. Dr. med. W. Klosterkötter) im Universitätsklinikum der Gesamthochschule Essen

Received July 28, 1975 / Accepted August 4, 1975

Summary. In two experimental series (19 33 nights, respectively, in 2 different persons in each series; testtime 10.30 p.m. to 1.00 a.m.) fingerpulse amplitudes after sonic booms were recorded during sleep. In the first 3 nights the subjects slept undisturbed by noise. In the following 11 and 10 nights, respectively, sonic booms were applied alternately 2 or 4 times. In the main series, after 10 more nights without noise, 4 nights with 8 and 16 sonic booms alternately followed. The last 5 undisturbed nights in both series were used as a comparison phase. The interval between two sonic booms was 40 min in nights with 2 booms, 20 min in nights with 4 booms and in the nights with 8 and 16 sonic booms 6.6 and 4.6 min, respectively. Sound level of the sonic booms ranged from 0.48 to 1.45 mbar; 1 mbar (83.5 dB[A]) on the average. The first sonic boom was applied if one of the two subjects had entered the deepest stage of sleep.

Sonic booms induced a decrease in fingerpulse amplitudes, which begin 3 sec after the onset of the stimulus. The minimum occurs after 6 sec, followed by an increase towards the preboom value. The reduction of the fingerpulse amplitudes is very significant (P < 0.01) from the 4th to the 32nd sec. This reaction was analyzed with special regard to the following factors:

1. Intensity: Due to the very short rise time of noise intensity there was no significant correlation between the maximum intensity of each boom and the decrease of fingerpulse amplitudes.
2. Hemodynamic variables: There are no significant connections between postboom fingerpulse amplitudes and noiseless time before the sonic boom, the duration of the test series, and the ambient temperature.
3. Endocrine variables: No correlation could be found between the stage of sleep and the reaction. On the contrary, a very significant correla-
ation was found between the reduction of fingerpulse amplitudes and the pulse rate before boom. With increasing pulse rate the extent of reaction becomes smaller.

Key words: Sleep - Sonic boom - Moderator variables - Fingerpulse amplitudes.

INTRODUCTION

Different reactions within the human organism are induced by acoustical stimulation. The vegetative centers of the brain stem are stimulated via the vestibulocochlear nerve and the formatio reticularis. The resulting ergotropic reactions are, for instance: increase of respiration frequency, peripheral resistance, and metabolic rate, decrease of cardic output, hand volume, and hand temperature as well as dilatation of the pupils. Thus, application of noise leads to numerous reactions of different vegetative functions. The extent of these reactions is dependent on intensity and hand width (Corbeille & Balder, 1929; Lehmann & Tamm, 1956; Oppinger & Grandjean, 1959; Jansen & Rey, 1962; Damsky, 1965).

One of the most simple indicators (in application and evaluation) of these ergotropic changes is the recording of fingerpulse amplitudes (FFA). Acoustical stimuli induce a great decrease of amplitudes ("on-reaction"), which is - when stimulation remains - followed by a steady value beneath the initial value. As a rule fingerpulse amplitudes begin to increase toward the initial value after the end of stimulation.

Because of the fact that acoustical stimuli cause alterations of the vegetative situation, these reactions consequently must be dependent on the vegetative situation immediately before stimulation. Due to the law of initial value (Wilder, 1931) the ergotropic reaction becomes smaller with increasing ergotropy. This law had been proved in several noise investigations such as, for instance, by Heinacker & Zipf (1960) as well as by Griefahn (1974), who recorded the changes of fingerpulse amplitudes.

Short noise exposures only induce on-reactions. In the investigation described here the alterations of fingerpulse amplitudes due to sonic booms (300 msec) had been proved as well as their dependency on the different moderator variables (intensity of sonic boom, quiet time before sonic boom, duration of test series, ambient temperature, sleep stage, and pulse rate).
evaluation of sleep stages

--- stage of sleep 0-1
--- stage of sleep 1
--- stage of sleep 1-12
--- stage of sleep II
--- stage of sleep II-112
---/ stage of sleep III

Fig. 2. Evaluation scheme of sonic boom experiments

**METHOD**

Fig. 1 presents a survey of the arrangement and the duration of both test series as well as of the recorded physiologic and physical parameters. Two subjects slept in the test room which had an opening to the sonic boom channel (SRL report).

During the entire test time (10.30 p.m. - 3.00 a.m.) EEG and fingerpulse amplitudes (FPA) were recorded continuously. The first sonic boom was applied as soon as one of the two subjects had reached the deepest sleep stage and the other subject had reached at least stage I. The pressure of the sonic booms applied in this investigation had the typical N-shape, the duration was 300 msec, the intensity ranged from 0.48-1.43 mbar, 1 mbar on the average.
EVALUATION

Because of several artifacts in the curves (body movements, etc.) only 261 single tests were evaluated. This evaluation was done in the following way:

Sleep Stages. Evaluation of sleep stages according to Fig. 2; the sleep stage within the minute before stimulation was considered an endogenic moderator.

Preboom Value of the FPA. Determination of the maximum amplitudes of the last 10 pulse waves preceding the sonic boom with an accuracy of 0.1 mm.

Postboom Value of the FPA. Until the 30th postboom sec the maximum amplitude of each single pulse wave and its distance to stimulus onset was measured. Related to the preboom average the maximum amplitudes were converted into percentage values. The distances from stimulus onset up to the maximum amplitudes (measured in mm) had been converted into seconds. These distances had been classified into 0.2-sec-distances; each amplitude within the time 1.31 and 1.50 sec had been considered as if it had occurred at 1.4 sec.

STATISTICS

Calculation of averages and standard deviations in 0.2-sec- or 1.0-sec-distances.

Calculation of the FPA parameters out of each single curve (Table 1).

Calculation of the correlation coefficients between FPA parameters and intensity as well as all other exogenic and endogenic moderator variables.

Calculation of the partial correlation with exclusion of the five other influences.

Calculated values were considered as significant only if p = 0.05. Because of the very small number of subjects (2 in each test) it was necessary to prove those significances for several subgroups (each subject of the main test in nights with 2 and 4 booms, in nights with 8 and 16 booms, and for the subjects of the pilot study). Only if these values were considered significant, also was a value described as significant in this study.

RESULTS

Fig. 1 demonstrates the course of the fingerpulse amplitudes after a sonic boom. Three sec after stimulus onset amplitudes
Table 1
Parameters calculated from each single curve and moderat variables

1) FPA parameters
- A = distance between onset of stimulus and minimum of fingerpulse amplitudes (sec)
- B = minimum of fingerpulse amplitudes (%)
- C = 1-sec-value of fingerpulse amplitude
- D = 10-sec-value of fingerpulse amplitude
- E = 22-sec-value of fingerpulse amplitude
- F = ascent between 3-sec-value and minimum
- G = ascent between minimum and 22-sec value
- H = angle above the minimum
- I = integral (beneath 100% value)
- J = average of fingerpulse amplitudes (4-22 sec in %)
- K = standard deviation (%) of deviation from 100% value without consideration of the direction (%)

2) Moderator variables
- M = intensity of sonic boom (mbar)
- N = quiet time before sonic boom (min)
- O = experimental night
- P = ambient temperature (°C)
- Q = sleep stage (preboom)
- R = preboom pulse rate

Fig. 3
Decrease of fingerpulse amplitudes after sonic booms during sleep

Sonic boom intensity 246-413 mbar (1-6 mbar), duration 300 ms,
1 or 10 sonic booms/night,
1 trial between 2 sonic booms 20 min, 20 min, 40 min, 60 min,
1-28 steps 5-15 (Stepwise, 15 trials each step)
4 subjects 645 2 sonic booms.
Fig. 4. Decrease of finger pulse amplitudes after sonic booms due to different preboom values

begin to decrease; the minimum occurs after 8 sec followed by an increase towards the preboom value. After the 22nd sec the decrease of amplitudes is no longer significant (P = 0.1%), so that all other calculations were done from the 4th until the 22nd sec.

The minimum duration of the pulse waves was 0.57 sec, so that at least 3 successive values of the demonstrated curve (distance 0.2 sec) were calculated from different experiments.

1. Intensity
Between FPA parameters and intensity of sonic booms (0.48–1.41 mbar) no correlation was found.

2. Exogenic Moderators
There was no significant connection between FPA parameters and exogenous influences [interval between sonic booms (quiet time before stimulus), duration of test series, ambient temperature]. For one single experimental night it was not possible to calculate significant correlations.

3. Endogenic Moderators
The sleep stages had no influence on the extent of the reaction.
Very significant was the connection between the preboom pulse rate and the FPA parameters C, D, and E [14-, 18-, and 12-sec values, coefficients (partial): 0.340, 0.351, 0.292, df = 256].

Therefore the curves of FPA after sonic booms were presented in Fig. 4 due to the level of pulse rate before stimulation (SP). The decrease of amplitudes is greater with decreasing pulse rates.

DISCUSSION

Three sec after the onset of sonic booms, fingerpulse amplitudes begin to decrease. The minimum occurs after 8 sec, followed by an increase towards the preboom value. The reduction of the amplitudes is significant (P < 0.01) from the 14th to the 22nd sec. The small deviation of the succeeding values gives evidence of the high reliability of these results.

1. Intensity

As pointed out by Jansen & Rey (1962) the decrease of fingerpulse amplitudes becomes greater with increasing intensity as well as with increasing bandwidth. According to an increasing number of stimulated nerve fibers that means with an increasing number of impulses per second a greater number of impulses arrive at the vegetative centers in the brain stem causing a stronger reaction. The missed relation between the FPA parameters and the boom intensities in this study may be caused by different factors. As described by Miederhoff (1974) the on-reaction becomes greater and occurs earlier when the rise time of noise pressure decreases. The rise time of the sonic boom is extremely short (5 msec in average) so that the organism may be unable to respond adequately. On the other hand, the range of intensities (80-89 dB(A)) may be too small to cause different reactions (Ludlow & Morgan, 1972). As pointed out by Jansen (1967) the increase or reaction becomes smaller with increasing intensities; the minimum occurs within the range of intensities used in this study.

2. Exogenous Moderator Variables

Quiet Time before Sonic Booms. The relation between the quiet time before sonic booms and the extent of FPA decrease was not significant. This corresponds to the results of Jansen (1974), who applied noises with increasing intensity (ambient noise level - 105 dB[A] within 15 sec) and increasing frequency. These noises, applied at intervals of at least 8 min always
caused the same reaction, whereas an application in less than 5 min caused a smaller reaction, indicating that the initial value is not yet regained. In this study, however, some booms with intervals up to 4 min always caused the same reaction. That fact points out that recovery needs less time with increasing vagotomy.

Duration of Test Series. Between the duration of the test series and the noise-induced decrease of fingerpulse amplitudes it was not possible to calculate any correlation. These results are in accordance to the results of McDonald et al. (1964) and Johnson et al. (1965), who described a decreasing ability of adaptation in drowsy and sleeping subjects. On the other hand, the intensities applied in this investigation are within a range which only causes defensive reactions, that is, reactions independent from habituation (Sokoloff, 1963). This independence could be proved not only for several months but also for several years. Jansen (1971) tested 9 subjects within 2 months, whereas Lehmann & Tamm (1956) examined the reaction of noise workers and students. None of these investigations gave evidence of any adaptation.

Ambient Temperature. Examinations of noise-induced decrease of fingerpulse amplitudes during different levels of ambient temperature have been done by Jansen (1967). Increasing temperature causes a vasodilation so that the same noise becomes less effective. During an ambient level of 40°C and a 95 dB white noise, no reaction could be recorded. In the study described here the range of temperature was possibly too small (5.5°C in the pilot study 6.5°C in the main study) to cause different reactions.

3. Endogenic Moderator Variables

Sleep Stages. In spite of the fact that an increasing depth of sleep is accompanied by an increasing vagotomy (Hess, 1933) no correlation could be calculated between the sleep stages and the FPA parameters. Williams et al. (1964), also, could not find any dependency of peripheral vasconstriction on sleep depth.

The variation of the vegetative situation during sleep is probably not important enough to cause different reactions.

Preboom Pulse Rate. The connection between preboom pulse rate and FPA-parameters (C, D, and E 14-, 19-, and 22-sec value) was highly significant. With increasing preboom, pulse rate reaction decreases. These results are in accordance with the
law of initial value (Wilder, 1931). This law, which has been proved in several investigations, demonstrates that the ergotropic reactions become smaller with increasing endocrin.

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SECTION 9
### Summary Form for Studies on the Effects of Noise on the Cardiovascular System (CVS)

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>W. Havel</td>
<td>Institute for Applied Physical Science, University of Dortmund, Abteilung Physiologie, 4600 Dortmund, Koenigstr. 67, West Germany</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Investigator's Phone No.</th>
<th>Sponsoring Organization</th>
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<table>
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<tbody>
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<td>28</td>
<td>8</td>
<td></td>
</tr>
</tbody>
</table>

**Purpose for study:** To study the relationship between performance, pulse rate, catecholamine excretion, and noise.

**Type & duration of experiment:** 10 weeks; one 4-hour session per week.

**Description of test groups (subjects, etc.):** 10 male college students, who served as their own controls; sessions 1-4, 6-9 - intermittent noise given and Kraepelin-Pauli tests taken; sessions 5, 10 - control sessions, no noise, no Kraepelin-Pauli tests.

**Control of other stresses:** Subjects were subjected to both noise and the performance test.

**Noise stimulus:**
- Source: not specified
- Spectral characteristics: pink noise
- Noise level: 90 dB
- Length of exposure: intermittent (30% of 70% quiet)
- # of trials: 10 (4 noise & 6 quiet)

**Statistical methods:** Factor analysis; covariance analysis

**CVS Response Measured:** Pulse rate

**Nonauditory effects:**
- CVS: pulse rate - increased due to noise
- Other: anxiety - decreased with noise
- Catecholamine excretion - increased due to noise
- Task performance - no different due to noise

**Author's conclusions:** No effects of noise on performance of the task was observed, due to the relative easiness.

**Evaluation & comments:**
2. Subjects were not studied with noise alone - but were given both a performance test and noise together (C stressor).

The relationship between task performance, intermittent pink noise, pulse rate, catecholamine excretion (adrenalin, noradrenalin) and anxiety was studied in ten male college students. Each student was tested with various combinations of noise, task performance, and quiet during one four-hour session per week for ten weeks. Pulse rate, catecholamine excretion, and psychological state were monitored for each session. The subjects were given both intermittent noise and intellectual tests in four of the weekly sessions. In another four sessions, the subjects took the tests under quiet conditions. The control sessions in the 5th and 10th weeks, consisted of no noise and no test-taking during which the students could study on their own. The 90 dB pink noise was given intermittently 30-70% of the session. No effects of noise on the performance of the test, which consisted of simple computations, were noted. The effects of noise included increased pulse rate and catecholamine excretion, and decreased anxiety.
RESEARCH ON THE PSYCHOLOGICAL AND PSYCHOPHYSIOLOGICAL EFFECTS
OF REPEATED, FOUR-HOUR INTERMITTENT PINK NOISE.

Dedicated to the honor of Prof. Dr. med. Dr. med. h.c.
Gunther Lehmann.

Zeitschrift für experimentelle und angewandte Psychologie

From the former Max-Planck Institute for Work Physiology
Dortmund. Former director: Prof. Dr. h.c. G. Lehmann.
Psychological Department. Former director: Prof. Dr. J.
Rutenfranz and the Institute for Work Physiology at the
University of Dortmund---Department of Environmental
Physiology---Director Prof. dr. med. H.C. Wenzel.

By Wolfgang Hauel

Ten male test personnel (students of the Dortmund
institute) took part in a test series lasting for ten
weeks, one test per week, on four hour sittings. In each
session they could work freely at least two hours for
their study. In the sessions 1-4 and 6-9, they had to
carry out the Krapelin-Pauli work test and alternating
from session to session, they were subjected to periodically
interrupted pink noise (90 dB (lin), 5 sec, 30% on,
70% off) and then to silence. The pulse frequency was
recorded during the Krapelin-Pauli test. Sessions 5 and
10 were control tests without noise and without Krapelin-
Pauli tests. The personality variables in question and
the catecholamine precipitation were studied in all sessions.

The Krapelin-Pauli work test alone gave no indica-
tion of a noise effect, which was interpreted as a result
of the slight difficulty of the tasks. During the free
activity, the test persons got the impression of being able
to work very fast, but with much less concentration than
without the noise. Both test conditions without noise
appeared much more similar to the test persons than each
of these of the noise condition. The influence of the
noise on disposition was considerable. There were less
anxiety symptoms with noise than during quiet.

There were indications for a relationship between
performance, error frequency, pulse frequency and noise.
The study was carried out and promoted by the assistance of the German Research Society (AZ:Hz 400/6).
A. Korte and A. Hubert assisted in carrying out and evaluating the test.
The EDV was carried out on the IBM 360/44 of the Max Planck Institute for nutritional physiology (Director: Prof. Dr. Senno Hans).

Test setup for determining the catecholamine precipitation amid noise.
The scheme shows the test plan: left, from the top downward, the numbers of the test persons, to the right the days of the week on which they took part in the tests as a rule. Above, the numbers of the sessions, of which the first four can be called acclimatization, not included in the evaluation. The symbols o, - and + in the fields correspond to the following: o = without the Kraepelin-Pauli test, without noise, - = with the Kraepelin-Pauli test, without noise and + = with the Kraepelin-Pauli test, but with noise. It is recognized at once that test type o was used in sessions 5 and 10 while both other test types were arranged in such a way that each test person went through each test type one time, and that each of the two test types took place five times in each session number. The course of the different test types is evident from the lower part of the scheme.

A report is made concerning the psychological results of a study whose physiological findings are given elsewhere (Hawel and Starlinger, 1967). A detailed foundation of the test and a thorough description of the test plan and of the test conditions took place there. For this reason the test arrangement is only briefly reported here:

Ten male test persons (students of the Dortmund technical school) took part in a test against a measured recovery in ten successive weeks, mostly on the same day of the week, at the same time of day, for four hours (see Fig. 1). The test conditions varied between "with noise" and "without noise" as well as between "with the Kraepelin-Pauli work test" and as a control condition "without the Kraepelin-Pauli test". These control conditions were without noise and always fell in sessions 5 and 10. In the other sessions, actually one test person was subjected to noise, while the other carried out the test in quiet. The noise was periodically interrupted pink noise of 90 db (lin) (re 2·10⁻⁴ μb). In the first session 'with noise', there was street noise with decibel peaks from 85 db (lin). At 5 second cycle time, the noise was interrupted by 30% (seating 2-4) or 70% (seating 6-9) quiet (with reference to the considerations of Teichner et al.). The first four sessions were not taken into account as acclimatization in the physiological study. For the last six sessions we determined an increased adrenalin precipitation during noise in the second half of the session with a 9% probability of error. Also, a tendency toward higher pulse frequency during noise had to be assumed during the Kraepelin-Pauli test.
In weighing a possible psychological effect of long-lasting noise, we posed four questions for ourselves, for the answering of which the study should bring information.

1. Is there an influence on carrying out a simple performance test?

This question was studied in tests of shorter length (among others by Sanders, 1961; Lienert and Jansen, 1964).

2. How will actual performance be judged with the usual intellectual work?

3. Do mood and/or self-image of the test personnel change?

4. Is there a tendency toward anxiety? To study this question we introduced the activation hypothesis discussed in connection with the effect of noise on man (Hörmann and Todt, 1960; Schönpflug, 1969). According to it, it appeared not to be ruled out that long-lasting noise caused stimulation which can be accompanied by a tendency toward anxiety behavior (see also Lidberg and Lavi, 1969).

1. Performance Test

The Kraepelin-Pauli work test was selected (Pauli-arnold, 1961) in order to obtain data concerning the objective performance capability under both test conditions "-" = "without" and "+" = "with noise". We were presented with a relatively simple task (to add actually two one digit numbers during one hour), which can be arbitrarily repeated and about which a large number of tests prevail (Ulich, 1958; other literature in the case of Christiansen, 1961). The control test without Kraepelin-Pauli and without noise received the symbol "o".

The commands for the Kraepelin-Pauli test ("attention, started!", every three minutes "line!" and at the end "attention" "pencils up") were spoken on a tape and were given over the loudspeaker into the booth. In the case of the sessions with noise, they were superimposed. The test persons were questioned before each test performance— with a short specimen throughput—whether the commands arrived clearly. (The communication test leader-test person took place over a telephone installation). Their sameness over all the tests was guaranteed by the type of command giving. Each of both used tapes was played 40 times, and its length could be neglected at the end of the test series.
The evaluation of the data from this test (sessions 6, 7, 8 and 9) by means of the covariance analysis showed no difference. (The five values from the individual work curves 'without noise' "−" were taken as independent variables and brought into relationship with the corresponding values of the working curves 'with noise' ("+"). Actually, two successive time curves were correlated).

The comparison of the five derived values, total, error percentage, improvement percent, climbing height and peak time—by means of discrimination analysis (Faber and Nollau) gave a difference at the 10% level in the acclimatization phase (sessions 1 to 4), which however could no longer be observed in sessions 6-9. Even a T2 test for dependent random tests gave no difference. The mean values are shown in Table 1. The "climbing height" proved to be a problematical measure, because occasionally it was clearly a "sinking depth".

Table 1

The table shows the mean value vectors of the Kraemelin-Pauli work test. To the far left the sessions and beside them the noise conditions about which we are actually reporting.

| Sessions Noise Total Error Improvement Climbing Peak |
|------------------|-----------|-------------|-------------|-------------|
|                  | with 3196 | 4.8 8.0     | 46.6 5.7    |
|                  | 1-4       |             |             |
|                  | out 3186  | 6.1 5.2     | 35.3 10.4   |
|                  | w.o. 3623 | 7.2 5.4     | 43.3 7.3    |
|                  | 6-9       |             |             |
|                  | with 3640 | 5.7 4.9     | 48.7 8.3    |

2. Subjective Judgment of one's own Efficiency

As is thoroughly established in our report concerning the physiological results of our study, the test personnel could study during a part of the available test time. The idea of this was that for the test personnel, the participation in this test series became a part of their overall day and thus lost the character of an exceptional situation. In order to obtain information concerning the subjective judgment of one's own efficiency, at the end of this work, the test personnel filled out a questionnaire in which they entered what they had done, how long, and so on. They then judged their.
1. working tempo between counter parts
   fast oo oo very slow
2. the intensity of their work between
   concentrated oo oo un concentrations
3. their quality, between
   good oo oo poor.

The activities named by the test persons were grouped according to appearance. The result was six main groups in which the other categories were classified. Table 2 shows the result of this last step. [Cand. phil. E. Rützel worked with the evaluation of this part].

Table 2  Total hours for the three test conditions among which the different works for the study were carried out.

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
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<tbody>
<tr>
<td>ions rel. signs</td>
<td>fields</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>'A'</td>
<td>19.3</td>
<td>20.0</td>
<td>12.7</td>
<td>5.25</td>
<td>57.25</td>
<td>2.0</td>
</tr>
<tr>
<td>'B'</td>
<td>25.5</td>
<td>21.5</td>
<td>11.5</td>
<td>2.7</td>
<td>61.2</td>
<td>1.0</td>
</tr>
<tr>
<td>'C'</td>
<td>22.25</td>
<td>22.45</td>
<td>14.0</td>
<td>5.0</td>
<td>62.7</td>
<td>1.5</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pauli</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Test instructions, performance of the test, urine emission, idle times.

100.85
400.00

The sum of the hours is actually entered in the cells during which a certain activity was carried out during a certain test condition. To the total of these times (= 189.15 hours) there are also 100 hours for the Kraepelin-Pauli test and 110.65 hours for the test instructions, performance of the test, urine emission and idle times.

Only the categories A, B, C and D were considered with the further evaluation, because category 'E' (= personal) did not fall in the area of questioning and category 'F' (= technical sign) required mental activity in another way than the other categories. It is noteworthy however that both these categories are occupied under the noise condition higher or alone.

The subjective judgment of our own activities during the test was therefore studied only for four categories. We proceeded in such a way that slow-fast, concentrated-unconcentrated, and good-bad were entered in each of the six classes (scale points) of the three evaluation scales in the number of hours in which the four activities (A, B, C and D) were subjectively carried out during the three test conditions with each of these (quality) classes.
Table 3

|       | 1   | 2   | 3   | 4   | 5   | 6   | Summe |  |
|-------|-----|-----|-----|-----|-----|-----|-------| |
| I. langsam:+ | 2,25| 13,50| 14,20| 21,00| 3,90| 1,60| 57,25 | |
|       | -1  | 4,75| 7,25| 10,50| 14,20| 10,50| 31,50 | |
| I. konzentriert | 5,50| 10,10| 16,00| 9,85| 8,25| 3,00| 62,70 | |
| II. konzentriert | 0,60| 17,20| 15,50| 11,75| 10,00| 2,00| 57,25 | |
|       | -1  | 12,50| 20,25| 0,35| 14,70| 5,00| 61,20 | |
|       | +   | 11,50| 22,70| 17,50| 5,00| 3,00| 5,00| 62,70 | |
| III. gut | +   | 17,20| 19,25| 16,20| 11,50| 5,00| 4,00| 57,25 | |
|       | -1  | 12,50| 17,50| 20,50| 7,60| 4,50| 61,20 | |
|       | +   | 12,50| 22,25| 21,85| 6,00| 62,70 | |

(1) Total; (2) slow; (3) concentrated; (4) good; (5) fast; (6) unconcentrated; (7) poor.

Table 3 shows the compilation of these data: left the three scales or the three test conditions, above the six quality classifications, right the line totals.

In the following we now compare the distributions of the work hours in the quality classes of the three polarities, between the three test conditions by means of a 2x6 field chi-square test. The result was thus three (I, II and III) times three ("o", "-", "+"") chi square values (Table 4), with actually five degrees of freedom.

The subjective judgment of one's own activity between the three test conditions was not different at the polarity I 'slow-fast'.

At polarity II there was a difference between all three test conditions 'concentrated-unconcentrated'. This difference was the slightest between both conditions 'without noise' ('"o"' and "-"').

The polarity III, 'good-poor' gave no difference of the subjective judgment of one's own activity between the test condition 'with noise' ('"+"') and each of the two other conditions ('"o"' or "-"'), but not between them (both were without noise).
Table 4

I. "slow-fast"

<table>
<thead>
<tr>
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<tr>
<td>$+$</td>
<td>10.74</td>
<td>.10</td>
<td>2.52</td>
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<td>$-$</td>
<td>8.06</td>
<td>.20</td>
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II. "concentrated-unconcentrated"

<table>
<thead>
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<tr>
<td>$+$</td>
<td>19.93</td>
<td>.01</td>
<td>14.4</td>
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<tr>
<td>$-$</td>
<td>16.16</td>
<td>.16</td>
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III. "good-poor"

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<th>$p$</th>
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<td>$+$</td>
<td>19.76</td>
<td>.01</td>
<td>4.64</td>
<td>.50</td>
</tr>
<tr>
<td>$-$</td>
<td>13.96</td>
<td>.025</td>
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Table 5

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<thead>
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<th>good/poor</th>
<th>conc/uncon.</th>
<th>rapid/slow</th>
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<td>$+$</td>
<td>1.8</td>
<td>1.4</td>
<td>0.9</td>
</tr>
<tr>
<td>$-$</td>
<td>4.6</td>
<td>2.1</td>
<td>0.9</td>
</tr>
<tr>
<td>$o$</td>
<td>9.4</td>
<td>4.7</td>
<td>0.5</td>
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Table 6.--Multidimensional scaling of the polarity polarity profiles.

<table>
<thead>
<tr>
<th>Control 'o'</th>
<th>Mood before the test</th>
<th>Mood after the test</th>
<th>Situation after the test</th>
<th>Self after the test</th>
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<tbody>
<tr>
<td>without ' - '</td>
<td>Mood before the test</td>
<td>Self before the test</td>
<td>Mood after the test</td>
<td>Situation after the test</td>
</tr>
<tr>
<td>with ' + '</td>
<td>Mood before the test</td>
<td>Self before the test</td>
<td>Mood after the test</td>
<td>Situation after the test</td>
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</tbody>
</table>

Table 7.--Factor analysis. The table shows the variables in their sequence during the tests. The numbers (1) and (2) mean the first and the second session halves. The second session half began with the KRAEPELIN-PAULI working test. The charges are shortened to two places and multiplied by one hundred. Charges < ±0.4 are omitted for easier orientation "o" means 'without' noise and 'm' means 'with' noise. (1) Communalities; (2) Similarity; (3) Mean values; (4) scatterings.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Kommunalität</th>
<th>Mittelwert</th>
<th>Streuung</th>
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<tbody>
<tr>
<td>Adrenalin (1)</td>
<td>43</td>
<td>95</td>
<td>72</td>
<td>83</td>
<td>82</td>
<td>72</td>
<td>.91</td>
</tr>
<tr>
<td>Noradrenalin (1)</td>
<td></td>
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<td></td>
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<tr>
<td>Pulse 4</td>
<td>90</td>
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<tr>
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<td>87</td>
<td>82</td>
<td></td>
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<tr>
<td>Noradrenalin (2)</td>
<td>2.3</td>
<td>1.9</td>
<td>2.1</td>
<td>1.9</td>
<td>1.6</td>
<td>1.6</td>
<td>1.5</td>
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</table>

Column square totals
The differences were there the largest throughout between the conditions "+" (with noise) and "o" (without noise and without Pauli); on the other hand, they were smallest between conditions "-" (without noise) and "o" (without noise and without Pauli).

If one summarizes the evaluation categories 1-3 and 4-6 and if one divides the first by the second sums, then one obtains Table 5. The ratio figures of this table show the tendency of a subjective judgment of the test persons about their own working performance better than Table 3.

1.8 times more working hours were spent under the conditions "+" with the subjective impression of good work performance than with poor working accomplishment. Under the conditions "-" and "o" the ratio increased to 4.6 and 9.5. The ratios in the case of 'concentrated-unconcentrated' were similar.

3. Self Judgment and Judgment of Mood

Since the polarity scheme according to Hofstätter proved itself to be a good usable method in several tests for the comprehension of situative components (Havel, 1964, 1969, 1970), it was also used for the study of this question.

At the start of the session, the test personnel judged their momentary mood according to the polarity profile. The same judgments were also made at the end of each session and there was an additional judgment of the test situation.

The two dimensions given in Table 6 gave a multi-dimensional scaling of the profile data (Havel, 1974), of which the first is immediately recognizable as the inner-outer dimension—relative to the subject. All self judgments and mood judgments of the control conditions ('o') lay here in the positive area, while all situation judgments and the mood judgments are under the both other test conditions in the negative area. The second dimension results from the position of the situation judgment, where both still conditions lie at the same place in the positive area and the noise conditions lie in the negative area. As is expected, the situation judgment 'with noise' lies next and the mood judgment at the end of the sessions 'with noise'.
Discrimination analyses of the five profile triples showed no differences for the three test conditions between the self-judgments.

The mood positions at the start differed only between both test conditions 'without noise' ('o' and '-' with 2% coincidence probability.

The situational conditions corresponding to the three situations were judged in such a way that differences existed only between the test 'with noise' ('+') and both other test types, but not between these (0.1% coincidence probability).

At the end of the sessions, the mood judgment of the control condition ('o') differed very clearly from the judgments under both other conditions (0.1% coincidence probability); these differed from each other only relatively little (5% coincidence probability).

4. Tendency toward Anxiety

For the study of this question, we experimentally used a German formulation with five parallel forms of the Scheier and Cattellian "Eight Parallel Form Objective Anxiety Scale" (1960) which was kindly made available to me by Prof. Dr. J. Fahrenbert (s.a. Beyme and Fahrenberg, 1966).

The questionnaires were answered by the test persons actually toward the end of sessions 5–9.

The form given in the fifth session was not taken into account in the evaluation. It was supposed to serve acclimatization to the method. Clearly less "fear symptoms" resulted from the effect of noise (such as "fear of bodily illness," "forgetfulness", "trouble with breathing", "itching on the skin", "cold arms and legs", "Nausea", etc) than without the effect of noise. If one uses the values of one comparison group to differentiate a difference (46 men, mean value 9.2; our mean values '+' = 7.9; '-' = 9.15) it is natural to assume that our test person found himself in a relatively tense state with the then existing increased general activity level which limited his tendency or even his capability for personal considerations compared with the tests without noise ('-') and the comparison group. One could also speak of a generalization of numbness.
Admittedly this is opposed to the findings referred to from Blau, who subjected his test persons with the performance of the psychological test to noise of 103 dB (lim) and found no effects other than 'sonatic complaints of specific anatomical location and description'.

5. Relationship between psychological and physiological Data

Finally, two factor analyses were reckoned for the data from the tests 'without' and 'with noise' from sessions 6-9 (PAPADO). The following were included as variables: adrenalin and noradrenalin precipitation from both session halves; from the Pauli test the last (5th) value of the double smoothed working curve, the error percent and the peak time point (peak position) as well as the mean pulse frequency during the last quarter hour of the test; from the anxiety scale the 'anxiety symptoms' and the 'emotional nature of the remark'. The results of the analysis are shown in Table 7 in such a way that the sequence of the variables corresponds to its time sequence. Because of the slight occupation with the analyses, only charges up to ±0.40 were interpreted. In order to make the differences of both analyses clear, it is practical here to regard the corresponding factors from both test conditions in common. The analysis 'with noise' is maximally approached to the analysis 'without noise' according to the principle given by Fischer and Roopert (Fehhardt). Because the catecholamine values from WP were lacking, they were not taken into account with the evaluation of the variables relevant here, in order to make the results comparable.

Factor 1 without noise gives the negative relationship between the performance of the Kraepelin-Pauli test and the naming of anxiety symptoms. The later the peak time point lay, the higher the final performance and the less anxiety symptoms were named. (That can be interpreted that men under quite conditions are less hypochondriac (fearful)). The performance maximum is reached more independently with noise and the level of performance seems to be related to the adrenalin precipitation in the first half of the session. Moreover, pulse frequency and naming of anxiety symptoms correlate here.

Factor 2 shows the high stability of the noradrenalin precipitation in both halves of the session under both test conditions. Under noise there is also a relationship between naming the anxiety symptoms and the peak time point.
Factor 3 exhibits only gradual differences under both test conditions. The relationship between "emotionality of the observations" pulse and mistakes exists clearly only without noise.

Factor 4 testifies of the proportionality of the adrenaline precipitation in both session halves and of the opposite tendency of the noradrenaline precipitation amid noise in the second session half.

Discussion of Results

1. In contrast to the studies of Sanders, but in agreement with Lienert and Jansen, we could find no influence of noise on the computing efficiency in the case of our performance test although a tendency to higher pulse frequency must be assumed during this test. The lack of performance difference is doubtlessly to be attributed to the small degree of difficulty of the task. The increased activation of the test persons during the noise sessions to be assumed on the basis of physiological findings could therefore only lead to another performance mode. Furthermore, it is to be considered that the test persons had absolved already before two four-hour sessions with noise and now in the sessions with noise, at the start of the performance test, were exposed to the same noise for two hours and supposedly had gotten used to the noise in this time.

   In contrast to these considerations, the possibility moved into the background the same performance moves thus that acoustical environment is created by the quiet as well as by the noise phase in which the minimum values in which the minimum values of the known, inversely U-shaped performance curve lie.

2. Comparatively opened up test arrangements—with free theme selection (so to say test person-centered or non-directive test arrangements in the mode of C.E. Rogers) with noise as stress are not known in the pertinent literature.

   The data concerning one's own working temp from which only slight differences result between the test conditions with noise and without noise, lie in the direction of the results from the Kraepelin-Pauli-test which is predominantly a speed test: hardly a finding, only a tendency to rapid work under noise.
Noise seems to have the greatest influence on the concentration, since the differences here are clearest compared with both other test conditions. But on the other hand, only the expectation of the Kraepelin-Pauli test appeared to have had a certain influence on the concentration of the test persons, for the tendency to unconcentrated work is noticeably greater under the concerned test conditions ("-") as over against the control sessions ("o"). Even the quality of the work is evaluated less amid noise than under both other test conditions.

In summary it can be said that in the subjective judgment under the noise conditions given here work was done with much less concentration and much worse than without noise, but that also a relatively short visit (as with the Kraepelin-Pauli test) had an influence on the concentration and quality of the performance.

3. The study of self-image, mood and test situation confirms the results as they were found with the other questions (Javel, 1964, 1969, 1970). So perhaps the constancy of the self-judgment and the agreement with mood judgment at the start of the tests with self-judgment or the influence of the test situation on mood judgment at the end of the sessions.

Profile comparisons show that under control (or quiet) conditions, the mood judgments lie completely with self judgments. Mood and self experience agree with each other; the situation was evaluated as something which is largely independent.

Under the condition with Kraepelin-Pauli, but without noise, the mood is largely influenced by the previous but already processed computation task.

Now under noise conditions, the test persons seemed to crawl into themselves at the start of the test, but later their mood is expressed by the computation task in connection with the noise, but very intensively by the situational conditions.

Both test conditions 'without noise' appear much more similar to the test persons than each of the two noise conditions.
4. Counter to our assumption that noise brings about a tendency to anxiety and to fear reactions, we found that there were no differences in the fear scale to be processed more cognitively and that during noise, less "fear symptoms" were named from the physical area. The assumption of a 'general numbness' used here as an interpretation aid in the sense of an irritation generalization presumably goes together with an ergotropic change of the vegetativum (Jansen, 1967).

5. The relationships existing here (factor 3) under both test conditions between error percent, emotionality of the remarks, and pulse frequency corresponds well to the Cattell-Schafer concept: "A refusal to deal seriously with the issue at hand." Admittedly, this relationship is more narrow without noise than with noise. Thus it can be said that during noise, with an increasing tendency of the pulse frequency, its relationship to the number of errors declines (and this itself), while a noticeable relationship to performance comes about, which is to be interpreted as the fact that at increased heart frequency, the performance maxima lie earlier and lower than with a slow heart frequency. Care is to be taken that the pulse frequency is registered only during the Knaepelin-Pauli work test. It is interesting that during noise, higher adding efficiency goes along with a tendency to low pulse frequency. In his studies, Rutenfrans found with the increasingly rapidly rotating Graf computer cylinder that the pulse frequency becomes higher and higher during the computations with more difficult tasks of the type $\text{W}_1\text{X}_1\text{Y}_1\text{Z}$ with increasing time pressure, but in the case of an easy task under the same conditions it fluctuated only slightly around a solid value. Because the performance motivation was well recognizable among the test persons, we may assume that the ones who found adding difficult had been under more stress, which resulted in our findings.


PAPADO: Dortmund Version of the Program PAPA of DRZ, Darmstadt.


Address of the author: Dipl. Phych. Wolfgang Holme, Institute for work physiology at the University of Dortmund, Environmental physiology department, 4600 Dortmund, Ardey St. 67. West Germany.
Summary

Ten male subjects (students) were required to take part in ten weekly sessions of four hours each. In the 5th and 10th session, the subjects did their own study work without any noise disturbance. In the second half of these main experimental sessions, they were required to do adding work (Kraepelin-Pauli-Test) every session under conditions of discontinuously presented noise of 90 dB (In). During the Kraepelin-Pauli-Test, the pulse rate was recorded. Relevant personality variables and corollary activation were studied in every session.

No effect of noise could be recorded on the Kraepelin-Pauli-Test, probably because this test is too easy. While doing their own study work, the subjects had the subjective impression of working more poorly with less concentration, but more fluently under the conditions of noise than in silence. Subjects found both the experimental situations rather similar. Noise had a great effect on the mood of the subject. The subjects reported more anxiety symptoms during the silence than during the noise.

Some relation between the noise and the performance, number of mistakes, and pulse rate could be determined.

Péryonie. Les auteurs discutent quelques observations, mais tentent une explication de leurs résultats et font le point quant à l'utilité de l'adaptation allemande du questionnaire de Stumphauzer.

Literatur


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2. Exp. Angew. Psychol. 22(4): 613–629

Zeitschrift für experimentelle und angewandte Psychologie
1972, Band XXII, Heft 4, S. 613–629

Research on the psychological and psychophysiological effects of repeated four-hour intermittent pink noise.

Aus dem ehemaligen Max-Planck-Institut für Arzneipflanzenbiologie Dortmund
Diplom-Direktor Prof. Dr. Dr. h. c. G. Gehrmann
– Psychologische Abteilung –
Diplom-Leiter Prof. Dr. Dr. h. c. weitere
und dem Institut für Arzneipflanzenbiologie in der Universität Dortmund
– Abteilung, Universitätspsychologie –
Doktor Prof. Dr. med. H. G. Wenzel

Untersuchungen zur psychologischen und psychophysiologischen Wirkung von wiederholtem vierstündigen, intermittierenden rosa Rauschen.1

Wolfgang Havel


Der Kraepelin-Pauli-Arbeitsversuch: Alle verkaufte keine Hinweise auf eine Lernrückwirkung, was als Folge der geringen Salvenzahl der Ausgaben gesehen werden kann. Während der freien Tätigkeit gemessen wurde, die Versuchsreihen der Einfluß der Lernvorschläge, was die erbrachte und konzentrierter und viel intensiver ausgeprägt war als kleines Lernen. Den Versuchspersonen wurden die hohen und hohen Verhaltensänderungen ohne Lernen sehr viel besser als eine dieser der Lernbedingungen. Der Einfluß des Lernens auf die Stimmung war erhöht, mit Lernen wurde eine geringere Verhaltensänderung genannt als bei Stille.

Es sind sich Anzeichen für eine Beziehung zwischen Leistung, Fehlerhäufigkeit, Pulsfrequenz und Lernen.

1) Herren Prof. Dr. med. Dr. med. h. c. Günther Lehmann zum Gedenken in Verbindung gewidmet.
Es wird über die psychologischen Ergebnisse einer Untersuchung berichtet, deren physiologische Bedingungen in einer anderen Arbeit mitgeteilt worden sind.

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Die EDV wurde an der IBM 360/44 des Max-Planck-Instituts für Ernährungsphysiologie (Dritter: Prof. Dr. F. Horn) durchgeführt.

Abbild. 1

Wolfgang Havel, Untersuchungen zur psychologischen und psychi-

Diese Frage war in Versuchen von kürzterer Dauer untersucht wor-

2. Wie wird die eigene Leistung bei gewohnter gleichartiger Arbeit beurteilt?
3. Ändern sich Stimmung und / oder Selbstbild der Versuchsper-sonen?
4. Zeigt sich eine Neigung zu Angstlichkeit? Zur Untersuchung dieser
Frage führten uns die u. a. in Verbindung mit der Wirkung von Lärm
auf den Menschen diskutierte Aktivierungshypothese (Hütermann &
Todts, 1965; SchünTEGRUPP, 1969). Nach ihrer erschien nicht auszu-
schließen, daß lange dauernder Lärm eine Erregung bewirke, die von
einer Neigung zu Angstlichen Verhaltensweisen begleitet sein kann

1. Leistungsversuch

Um Daten über die objektive Leistungsfähigkeit unter den beiden Ver-
suchsbedingungen, "— " = "ohne" und "+" = "mit Lärm" zu erhalten,
werden der KRAEPPEL-Pauli-Arbeitsversuch ausgewählt (Pauli —
Arnold, 1961). Er stellt eine relativ lange einfache Aufgabe dar
(10 Minuten, zwei einstellige Zahlen, während einer Stunde), die
sich beliebig wiederholen läßt und über die also eine große Anzahl von
Untersuchungen verfügen (Ullrich, 1964, weitere Literatur bei 
Christiansen, 1960). Der Kontroll-Versuch, ohne Kraepelin-Pauli und
ohne Lärm, erhielt das Symbol "\(\ast\)."

Die Kommandos für den Kraepelin-Pauli-Versuch (Achting, —
afang, +, alle 3 Minuten "Stich" und am Ende "Achting, Blau
stift weg") waren auf Send gesprochen worden und wurden über die
Lauftrager in die Kabine gegeben. Bei den Sitzungen mit Lärm wurden
sie doppelt überlagert. Die Versuchsperonen wurden jeweils vor jeder
Durchführung des Tests — bei einer kurzen Prüf-Durchsage — befugt,
ob die Kommandos auch deutlich anklingen. (Die Kommunikation Ver-
suchsleiter-Versuchsperon erfolgte über eine Telefonleitung.) Durch
diese Art der Kommando-Gabe wurde außerdem ihre Gleichheit über alle
Sitzungen gewährleistet. Jedes der beiden verwendeten Bänder wurde
4mal gespielt, und ihre Dehnung konnte auch am Ende der Versuchs-
tafel veranschlagt bleiben.

Die Auswertung der Daten aus diesem Versuch (Sitzungen 2, 7, 8 und
9) mittels Kovarianzanalyse ergab keinen Unterschied. (Die fünf Werte
aus den individuellen Arbeitskurven ohne Lärm (—) wurden als unab-
hängige Variablen genommen und in Beziehung zu den korrespondieren-
den Werten der Arbeitskurven mit Lärm (+) gebracht; dabei wurden
jeweils zwei zeitlich aufeinanderfolgende Kurven korreliert.)

physiologischen Wirkung von wiederholten Varianten usw. 617

Der Vergleich der fünf abgeleiteten Werte — Summe, Fehlerprozent,
Verbesserungs-Prozent, Steilhöhe und Gipfelhöhe — mittels Diskrimi-
nananalyse (Faber & Nollau) ergab in der Gewöhnungsphase (Sitz-
ungen 1 mit 4) einen Unterschied auf dem 15% Niveau, der aber in den
Sitzungen 6 mit 9 nicht mehr beobachtet werden konnte. Auch ein T"-Test
für abhängige Stichproben ergab keinen Unterschied. Die Mittelwerte

Tabelle 1

Die Tabelle zeigt Mittelwerte-Vektoren der Kraepelin-Pauli-Ar-
beitsversuche. Ganz links die Sitzungen und daneben die Lärmbedingun-
gen, über die jeweils gemittelt wurde.

| Sitzungen | Lärm | Summe | Fehler-Prozent | Verbesser-

Tagespunkt | Steil-

höhe | Gipfel-

höhe |
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Vektoren sind in Tabelle 1 dargestellt. Die "Steilhöhe" erwies sich als
proportionalen Maß, weil sie gelegentlich eindeutig eine "Sinktiefe" war,

2. Subjektive Beurteilung der eigenen Leistung

Wie in unserem Bericht über die physiologischen Ergebnisse dieser Un-
tersuchung ausführlich begründet, konnten die Versuchsperonen während
eines Teils der zur Verfügung stehenden Versuchszeit für ihr Studium
arbeiten. Es sollte damit erreicht werden, daß für die Versuchsperonen
die Teilnahme an dieser Versuchsreihe ein Teil ihres Alltags wurde
und damit ihnen eine Ausnahmesituation verleih. Um Information
über die subjektive Beurteilung der eigenen Leistung zu erhalten, sollten
die Versuchsperonen, nach Beendigung der Studien, jeweils einen
Fragebogen aus, in den sie eintrugen, womit sie sich, wie lange, beschäf-
tigt hatten. Sie beurteilten dann:

1. Ihr Arbeiten, zwischen dem Gegenübersprach
fein 00000 langsam;
2. die Intensität ihrer Arbeit, zwischen
konzentriert 00000 unkonzentriert;
3. deren Qualität, zwischen
gut 00000 schlecht.

Tabelle 2

<table>
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Tabelle 3 zeigt die Zusammensetzung dieser Daten links die drei Skalen bzw. drei Versuchsbedingungen, oben die sechs Gutakten, rechts die Zeilensummen. Im folgenden wurden nun die Verteilungen der Arbeitsstunden auf die Guteklassen der drei Polariitäten, zwischen den drei Versuchsbedingungen mittels eines 2 x 6-Felder Chi-Quadrat-Tests verglichen. Es ergaben sich somit drei (I, II und III) mal drei (o, +) Chi-Quadrat-Werte (Tabelle 4), bei jeweils fünf Freiheitsgraden.

Auf der Polariität I, langsam—fertig, war die subjektive Bewertung der eigenen Tätigkeit zwischen den drei Versuchsbedingungen nicht unter- | ständig. Auf der Polariität II, konzentriert—unkonzentriert, bestand zwischen allen drei Versuchsbedingungen ein Unterschied dabei war dieser Unter- | schied zwischen den beiden Bedingungen 'ohne Lern' (o+o) und 'mit Lern' (+) am geringsten. Die Polariität III, gut—schlecht, ergab einen Unterschied der subjek- | tiven Bewertung der eigenen Tätigkeit zwischen der Versuchsbedingung 'mit Lern' (++) und jeder der beiden anderen Bedingungen (o+o bzw. o—o) nicht aber zwischen diesen (beide waren ohne Lern).
subjektiven Urteile der Versuchspersonen über ihre eigene Arbeitsleistung.

Unter der Bedingung + wurden 1,5mal mehr Arbeitsstunden mit dem subjektiven Eindruck geringerer Arbeitsleistung verbracht als mit schlechter Arbeitsleistung unter den Bedingungen - und -; die Versuchspersonen bewerteten die Hörbeläge aber auf 4,6 bzw. 9,3 an. Ähnlich liegen die Verhältnisse bei konzentriert—unkonzentriert.

3. Selbstbeurteilung und Beurteilung der Stimmung

Da das Polaritäts-Schema noch als Hilfsmittel sich sehr in mehreren Untersuchungen als gut brauchbares Verfahren für das Erfassen situativer Komponenten erweisen hatte (Za e. a.), traf es sich auch für die Untersuchung dieser Frage Verwendung.

Zu Beginn jeder Sitzung hatten die Versuchspersonen sich selbst und ihre augenblickliche Stimmung nach dem Polaritäts-Profil zu beurteilen. Die gleichen Beurteilungen wurden auch am Ende jeder Sitzung vorgenommen und zusätzlich eine Beurteilung der Versuchssituation.

Tabelle 6
Mehrdimensionale Skalierung der Polaritätsprofile.

<table>
<thead>
<tr>
<th>Kontrolle</th>
<th>Stimmung vor dem Versuch</th>
<th>Selbst</th>
<th>Stimmung nach dem Versuch</th>
<th>Situation</th>
<th>Selbst</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td>0</td>
<td>26</td>
<td>37</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>-</td>
<td>0</td>
<td>37</td>
<td>32</td>
<td>7</td>
<td>0</td>
</tr>
</tbody>
</table>

mit Lärm

<table>
<thead>
<tr>
<th>Kontrolle</th>
<th>Stimmung vor dem Versuch</th>
<th>Selbst</th>
<th>Stimmung nach dem Versuch</th>
<th>Situation</th>
<th>Selbst</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td>-8</td>
<td>3</td>
<td>21</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>-</td>
<td>-3</td>
<td>66</td>
<td>65</td>
<td>14</td>
<td>0</td>
</tr>
</tbody>
</table>
Eine multidimensionale Skalierung der Profiltupfen (Ha weI, 1974) ergab in Tabelle 6 wieder gegebenen zwei Dimensionen, von denen die erste ohne weiteres als die — auf das Subjekt bezogene — Innen-Außen-Dimension erkannt wird. Alle Selbstbeurteilungen und die Stimmungsbeurteilungen der Kontrollbedingungen (A) liegen hier im positiven Bereich, während alle Situationsbeurteilungen von der Stimmungsbeurteilung unter den beiden übrigen Versuchspersonen im negativen Bereich liegen. Die zweite Dimension ergibt sich aus der Lage der Situationsbeurteilung, wobei die beiden Stimmungsbeurteilungen gleicher Stellung im positiven Bereich und alle Lürenbeurteilungen im negativen Bereich liegen. Der Situationsbeurteilung "mit Lärm" am nächsten liegt erwartungsgemäß die Stimmungsbeurteilung "mit Lärm".

Diskriminanzanalyse der fünf Profil-Tripels für die drei Versuchsbedingungen ergab zwischen den Selbstbeurteilungen keine Unterschiede.

Die Stimmungsplagen zu Beginn unterschieden sich lediglich zwischen den beiden Versuchsbedingungen "ohne Lärm" (A* und A**) mit 25% Zufallswahrscheinlichkeit.

Die den drei Bedingungen entsprechenden situativen Gegebenheiten wurden so bestimmt, daß sich allein zwischen dem Versuch "mit Lärm" (+) und dem beiden anderen Versuchsformen, aber nicht zwischen diesen, Unterschiede ergaben (je 0,1% Zufallswahrscheinlichkeit).

Am Ende der Sitzungen unterschied sich die Stimmungsbeurteilung der Kontrollbedingung (A*) sehr deutlich von den Beurteilungen unter den beiden anderen Bedingungen (8,7% Zufallswahrscheinlichkeit), die unterschieden sich voneinander aber nur relativ wenig (3,5% Zufallswahrscheinlichkeit).

4. Neigung zu Anreglichkeit

Für die Untersuchung dieser Frage wurde vorgezogen, eine deutsche Festung mit fünf Parallel-Formen der Scheler und Cattell'schen "Eight-Parallel-Form Objective Anxiety Scale" (1966) benutzt, die mit freundlicherweise von Herrn Prof. Dr. J. Fahrenberg zur Verfügung gestellt wurde (s. Beyer & Fahrenberg, 1966).

Die Fragebogen wurden von den Versuchspersonen je zwei gegen Ende der Sitzungen 5. bis 6. beantwortet.

Die in der fünften Sitzung gegebene Form wurde bei der Auswertung nicht berücksichtigt, so daß die Befragung an das Verfahren dienen. Es wurden unter Anreglichkeit deutlich weniger "Angst-Symptome" (wie "Furcht vor körperlicher Krankheit", "Vergänglichkeit", "Müde beim Atem", "Kribbeln in der Haut", "Kalte Glieder", "Dürsichtigkeit", etc.) angegeben als ohne Lärmwirkung. (Fisher, Pittman, p. 5, 015, a. Lennets). Wenn man zur Differenzierung dieses Unterschiedes Werte einer Vergleichsgruppe benutzt (46 Männern, Mittelwert 9,2; unsere Mittelwerte für .7" = 7.9, .6" = 9,3), liegt die Vermutung nahe, daß sich unsere Versuchspersonen während der Lärmverurteilung (+) bei dem dann gegebenen allgemeinen Aktivierungslevel, in einem verhältnismäßig angenehmen Zustand befinden, der ihre Neigung oder auch ihre Fähigkeit zur Selbstbeurteilung — verglichen mit den Versuchen ohne Lärm (—) und der Vergleichsgruppe — einschränkt. Man könnte auch von einer Generalisierung der Verzögerung sprechen.

Dies sei allerdings im Gegenworte zu den von Plutchik referiert Befunden Blau's, der seine Versuchspersonen bei der Durchführung von psychologischen Tests, Lärm von 103 db (B) auftrat und dabei keine Wirkungen fand als "sonnie complaints of specific anatomiak loadon and deretpnten".

5. Beziehung zwischen psychologischen und physiologischen Daten


Faktor 1 gibt ohne Lärm die negative Beziehung zwischen der Leistung im Krespein-Pauli-Versuch und der Nennung von Angstsymptomen wieder. Je später der Gipfelzeitpunkt liegt, um so höher ist die Emotionsleistung und um so weniger Angstsymptome werden genannt. (Das
Table 7

| Faktorenanalyse in der Tabelle sind die Varianzen in ihrer Auswirkung auf die Augenabstände. Die zweite Spalte enthielt die Variablen zur Bestimmung der Augenabstände. Die Datensätze wurden als Bilanzseite angeordnet, um die Verteilung der Variablen zu ermitteln.

<table>
<thead>
<tr>
<th>Faktor 1</th>
<th>Faktor 2</th>
<th>Faktor 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenalin (1)</td>
<td>62</td>
<td>72</td>
</tr>
<tr>
<td>Faktor 4</td>
<td>50.55</td>
<td>55.55</td>
</tr>
<tr>
<td>Adrenalin (2)</td>
<td>67.52</td>
<td>70</td>
</tr>
<tr>
<td>Spaltabstände</td>
<td>2.21</td>
<td>1.16</td>
</tr>
</tbody>
</table>

Die Tabelle zeigt die Ergebnisse der Faktorenanalyse, die die Varianzen der Augenabstände aufzeigt. Die Faktoren zeigten eine starke Korrelation und korrelierten signifikant mit den Augenabständen. Die Faktoren wurden als Bilanzseite angeordnet, um die Verteilung der Varianzen zu ermitteln.

Physiologischer Wirkung von wiederholtem visuellen 

kann geübt werden, dass Menschen unter Ruhebedingungen bei zunehmendem Leistungsniveau (d.h. etwa ihre maximale Leistung systematisch aufbauen) weniger hypoxämisch (nichtsdestoweniger) sind. Mit Lärm wird das Leistungsumfeld unabhängig erreicht und die Härte der Leistungskonstellation in Beziehung zur Adrenalinausscheidung in der ersten Sitzungsphase zu stellen. Zudem korreliert hier Pulsfrequenz und Nettzeit von Angstsymptomen.

Faktor 2 zeigt die hohe Stabilität der Norexinsäurespiegel in beiden Sitzungshälfte unter beiden Versuchsbedingungen. Unter Lärm besteht dagegen keine Beziehung zur Nettzeit von Angstsymptomen und zum Spaltabstand.

Faktor 3 zeigt unter beiden Versuchsbedingungen nur geringe Unterschiede auf. Der Zusammenhang zwischen „Emotionalität der Bemerkungen“ und Puls und Fehlern besteht deutlich nur mit Lärm.

Faktor 4 zeigt von der Proportionalität der Adrenalinproduktion in beiden Sitzungshälfte und von der entsprechenden Tendenz der Norexinsäurespiegel unter Lärm in der zweiten Sitzungshälfte.

Diskussion der Ergebnisse


2. Vergleicht man die Tatsachen mit freien Themen durch - besczisge, Versuchspersonen, zentrierte oder nicht-direktive-
Wolfgang Hartl, Untersuchungen zur psychologischen und psychi.

Versuchsansordnungen nach C. R. Rogers mit Lärm als Stressor sind in der einschlägigen Literatur nicht bekannt.

Die Angaben über das eigene Arbeitsleistungsverhalten von geringer Unterschiede zwischen den Versuchsbedingungen mit Lärm und ohne Lärm zu liefern, liegen in der Regel der Erwartung aus dem Kraepelin-Pauli-Verkauf, der ja wesentlich ein Speed-Test ist, kaum ein Befund, lediglich Tendenzen zu stoffwechsel Arbeiten unter Lärm.


Zusammenfassend läßt sich sagen, daß im subjektiven Urteil unter den hier geprüften Lärmbedingungen eher flott aber erheblich unkonzentrierter und viel schläfer gescheitert wurde als ohne Lärm, daß aber auch ein verhältnismäßig geringer Einfluß auf Konzentration und Güte der Leistung hatte.


Unter der Bedingung mit Kraepelin-Pauli, aber ohne Lärm, ist die Stimmung anscheinend in hohem Maße von den noch beherrschten bzw. schon bearbeiteten Rechenaufgaben beeinflußt.

Unter Lärmbedingung nun scheinen sich die Versuchspersonen zu Beginn der Versuche in sich selbst zu verlieren, aber später ist ihre Stimmung durch die Redenaufgaben in Verbindung mit dem Lärm doch sehr intensiv von den situativen Gegebenheiten geprägt.

Die beiden Versuchsbedingungen ohne Lärm erweisen den Versuchspersonen einander sehr viel schläfer als jede von beiden der Lärmbedingung.

4. Entgegen unserer Annahme, daß Lärm eine Neigung zu Angst-


PAPAD: Darstellung der Programme PAPA vom DBZ, Darmstadt.


## Summary Form for Studies on the Effects of Noise on the Cardiovascular System (CVS)

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>C. Jansen (Prof. Dr. med. Dr. phil.)</td>
<td>Universitatsklinikum Essen, 43 Essen 1, Hufeland Str. 55, Federal Republic of Germany</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Investigator's Phone No.</th>
<th>Sponsor's Organization</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Deutsche Forschungsgemeinschaft (DFG)</td>
</tr>
</tbody>
</table>

### Citation

<table>
<thead>
<tr>
<th>Type &amp; duration of experiment</th>
<th>Purpose for study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Community survey, audiometric testing, psychological testing, pilot study—Hamburg airport area. Main study—Munich airport area</td>
<td>The sociological, physiological, and psychological impact of aircraft noise on the population surrounding Munich Airport.</td>
</tr>
</tbody>
</table>

### Description of test groups (subjects, if used, etc.)
660 persons, 15–70 years old, living in the vicinity of Munich airport in the first social survey. The subjects involved in the physiological and audiometric experiments and tests included 375 persons from 21 to 60 years.

### Control of other stresses
No control—field studies

### Noise Stimulus
Source: aircraft noise

#### Spectral characteristics
Not given

#### Noise level
Not given—varies

#### Length of exposure
Varies

### Statistical Methods
- Correlation determination coefficients; regression analysis

### CVS Response Measured
- Heart rate
- Blood pressure; fingerprint amplitude

### Nonauditory effects
- Most distinctive reaction to noise—CVS; decreased fingerprint amplitude reaction; linear relationship found between increasing noise (noise levels & % of flyovers) and reactions in humans—such as blood pressure (increased; heart rate decreased); annoyance

### Author's conclusions
In general, no adaptation to aircraft noise was found. Aircraft noise cannot be ruled out as a risk factor in hypertension.

### Evaluation & comments
This is just a brief summary of results from earlier studies reported at the Dubrovnik congress in May 1973. (An interdisciplinary study on the effects of aircraft noise on man, B. Rohrmann et al.)

The results of a series of surveys on the effects of aircraft noise on people who live near large airports are reported in brief summary form. A pilot study was done near Hamburg airport, after which the main study was conducted near Munich airport in East Germany. The studies gathered demographic, sociological, psychological, and physiological data on over 600 people from 15 to 70 years old. Cardiovascular responses to aircraft noise included decreased fingerpulse amplitude, decreased heart rate, and increased blood pressure. These reactions may be related to the annoyance caused by the aircraft noise. The author concluded that there was no evidence of adaptation and aircraft noise cannot be ruled out as a risk factor in cardiovascular disease. A more detailed report of the studies is published in the Dubrovnik congress (May 1973) proceedings by Rohmann et al. and titled "An Interdisciplinary Study on the Effects of Aircraft Noise on Man."
Physiological Responses Due to Noise in Inhabitants around Munich Airport

Prof. Dr. med. Dr. phil. Gerd Jansen
Universitätsklinikum Essen
43 Essen 1, Nordrhein-Westfalen 52
Federal Republic of Germany

Summary: The Deutsche Forschungsgemeinschaft (DFG) initiated and sponsored an interdisciplinary research on aircraft noise effects on inhabitants around airports. A pilot study (around Hamburg airport) and a main study (around Munich airport) were conducted by acoustical, epidemiological, social scientific, psychological, physiological and medical sections of scientists. It was found out that, in general, there was no adaptation to aircraft noise. There is an existing linear relation between increasing noise stimuli (sum of noise exposure and number of flyovers) and human reactions such as complaints or annoyance. Thus DFG initiated then an interdisciplinary research including not only sociological and psychological, but also physiological impact of aircraft noise.

1. Scope

The effects of aircraft noise on men living around airports were studied by an interdisciplinary team which was sponsored by the Deutsche Forschungsgemeinschaft (DFG). The main study was conducted around Munich airport and the pilot study was done around Hamburg airport.

There are existing some other investigations in the world using survey techniques but they confined to mainly sociological aspects such as complaints or annoyance. Thus DFG initiated then an interdisciplinary research including not only sociological and psychological, but also physiological impact of aircraft noise.

2. Organisation of the study

The team of the project was composed of 6 sections:
- "Acoustics" (H.-O. Finke, R. Martin, PTB Braunschweig)
- "Medicine" (W. v. Kief, L. Horbach, H. Jörgens, Umweltmedizin Essen)
- "Organisation" (D. Hohmann, Uni Mannheim)
- "Psychology" (H. Gücker, H. Noll, Uni Martin, Uni Hohenheim)
- "Social Science" (W. Schleer, H. Schmitz, A. Schmida, Uni Hohenheim)
- "Work-physiology" (G. Jansen, Umweltmedizin Essen)

Each section tested the same subjects to collect data for an interdisciplinary analysis.

Preparing the whole study the organisational section first selected the human beings living around Munich airport according to the exposure to the noise levels and secondly in accordance with demographic criteria. The whole area was divided into 32 areas with different noise levels; these "clusters" were combined to 4 "cluster-maps".

660 persons from 15 to 70 years were tested in a first social scientific step. Interviews based upon standardized questionnaires were taken at the respondents homes. This interview had a collecting function in order to ask the people to follow the second step of our investigation in psychological, physiological and audiometric experiments and tests. The combinations of 327 subjects in a separate test station took about 2 hours for each person.

The third step contained the medical case history, clinical examination and experiments at the test station. This step took another 2 hours for each person. The fourth step was a consecutive one, it consisted of acoustical measurements (one measuring point for each cluster). The tested subjects in the first and in the second step (psychological and medical examinations) were restricted to 327 persons aging from 21 to 60 years.

The interviews of the sociological section were extended in a second part to 15% of the inhabitants of the clusters who had moved during the last 12 months preceding the study.

3. Results

3.1. Former publications

The major results of the whole DFG-study were already reported at the congress at Dubrovnik in May 1973. Another survey of the results was given at Inter-Noise (Copenhagen 1973). Especially the relations between acoustical parameters and noise reactions in human beings were presented at the Transportation Noise Symposium (Southampton, July 28-29, 1974). The detailed DFG Forschungsbericht is in print and publication is expected on February 15, 1975.
1.2. Main results of the whole study

According to the assumption of the complex (multimodal instead of monomodal) system of interdependent variables these "moderator variables" were being attributed and rearranged to the investigation of nervous reaction effects of different reaction situations. This concept of moderators led to an interdisciplinary analysis and synthesis based on an analysis of the single sections. Thus, it is useful to know first the results of single sections.

1.2.1 Social survey

By means of regression and correlation techniques the social science section tried to clarify the relationship between stimulus moderators and reactions and between the relative contribution of stimulus and moderator variables to the prediction of reactions. It was found out that the relationship between stimulus and reaction variables is by no means perfect ones; the highest correlations reached to $r = 0.50$.

This result means that only about 25% of the variability in reactions can be predicted by means of one stimulus variable alone so that a considerable amount of the whole reaction remains unexplained.

As in other aircraft noise studies "disturbance of communication (disturbance in conversation, in listening to radio, TV)" was the greatest one, whereas other ones like "perceived number of times of aircraft noise", "irritability by aircraft noise", "disturbance of tranquility", "number of subjects sleeping" "the number of subjects sleeping when asked for interconveniences" etc., they all had smaller correlations.

All these relations are linear. Curvilinear determination coefficients led only to an insignificant increase as compared to linear determination coefficients. Even when correlating more than one stimulus variable no other result could be found.

1.2.2 Psychophysiological experiments

The psychophysiological laboratory experiments were done by the psychologists and work-physiologists together. On one side we had the hypothesis of "adaptive coping" with aircraft noise. On the contrary we tried to find out a "defensive blocking" which assumes an interruption of information processing and physiological states of defensive reactions. As a consequence of frequent and intense aircraft noise. Therefore the investigation was done under the aspect of the "general activation theory" of the psychologist. Spitz and the "orienting" and "defensive" components.

Moreover it was assumed that damping or disturbance of the information input is in accordance to the "distraction theory". Further on it was expected, there were connections between aircraft noise stimuli and aircraft noise reaction by pumpers' characteristics.

In order to find out the characteristic noise reaction of the people investigated, the psychologists used personality tests, recognition techniques, memory tests, signal detecting tests, and together with the physiological reaction they registered the behaviour of vasomotoric and muscular activity which were continuously recorded in experimental situations with quietness and noise interchanging.

It was not possible to confirm the hypothesis of "adaptive coping" with aircraft noise. The physiological responses due to noise increased in all cases. In detail, we saw a contraction of the bloodvessels at the finger and at the temple, an increase in the electrical muscle activity, and a decrease of the heart rate. This complex reaction was called "defensive reaction" allowing the psychologists. One can conclude from this that there could be at least a blocking of information reception processes. These defensive reaction is correlated positively with the intensity and frequency of the aircraft noise ($r = 0.31$). It occurs especially with those persons who were characterized by a "low mobility", by "strong conservative tendencies" and by a "very high blood pressure".

Moreover we saw that the hearing acuity decreased with increasing aircraft noise exposure. But this result is statistically insignificant. Other aspects of human psychophysiological behaviour especially psychological behaviour were not so much affected by aircraft noise.

1.2.3 Medical investigation

The medical examinations were done separately from that of psychophysiological sections after another two weeks. The people were assessed by means of anamnese and examination of body containing the analysis of clinical status as well as experimental tests of vegetative functions.

The analysis of the medical data could not prove any cases of manifest illness which is due to aircraft noise. In physiological experiments systolic and diastolic blood pressure, heart rate, respiration rate and electrical muscle activity were recorded for 30 minutes. The subjects were submitted to quietness, mental arithmetic, continuous noise, and discontinuous noise. There was only a tendency of change in vegetative functions especially regarding the diastolic blood pressure. The medical scientists have the opinion that it cannot be excluded that aircraft noise is a "risk factor" for the generation of essential hypertension or the bloodvessels.
1.1. Interdisciplinary Interpretations

The different data from the single sections were integrated (N = 357) to an interdisciplinary analysis which resulted only in low intercorrelations of the sociological, psychological, and physiological variables towards aircraft noise effects.

Using an interdisciplinary set of sociological, psychological, and physiological moderators of the variability of the "social-psychological" is determined by them whereas another third is determined by the stimulus variables. By using so-called path models the scientists doing the interdisciplinary interpretations found chains within one path model containing the factors "dissatisfaction to noise", "age", "sex", "fear associations", "attention performance" connecting them with "annoyance and disturbance reaction", "defensive reaction", "diastolic blood pressure" and the dependence of all of them to the noise load.

With reference to the noise protection measures as they are defined in several countries like USA, Great Britain, West Germany, etc., the scientists doing the interdisciplinary analysis found out that outside of the areas confined by this protection values there is a considerable percentage of the population which is highly annoyed and influenced by aircraft noise.

But the regression lines of the "disturbance of communication", "disturbance of rest and recreation" and the "feeling of aircraft as a disturbing factor spontaneously mentioned" were linear regression lines. So there is no point which could be regarded as intolerable noise level. There is only an increasing number of people who feel annoyed and who are influenced physically by increasing of aircraft noise. So they conclude that the protection of aircraft noise is a problem for those producing noise and also for those distributing noise. They feel that it is a problem involving aspects of engineering as well as of policy.

2. Discussion of results from physiological standpoint

Already the pilot study around Hamburg airport showed and proved that the results of former physiological noise research need no basic correction. The experimental psychology results of the Hamburg pilot study (aircraft noise, traffic noise and artificial white noise were applied), showed that the results were comparable to those expected from results of former noise research.

In the main study around Munich airport we tried to find out moderating factors of the physiological responses. These could give explanation of the value of the psychophysiological noise reaction within the total load of environmental factors of the human being. We stated already that the theory of "adaptive coping" had to be cancelled in favour of the "defensive reaction". The combined defensive reaction consisting of chest volume, muscle activity and tracking test, were regularly influenced by single noise bursts. Though the whole defensive reaction is correlated in a linear regression to the combined noise measure FDI (which contains the number of movements and the noise levels of the single movements) similar to the English NNI (noises number index) we saw the most distinctive reaction in the finger pulse amplitudes. Comparing these results with former investigations done with approaching aircraft noise and with the noise reactions of people with different personality moderator variables we think that the physiological measuring parameters are closer correlated to noise intensity level whereas the combined reaction in the physical as well as in the psychological behaviour is more related to the combined noise exposure measure (number of movements and intensity level).

This leads us to the conclusion that for noise assessments around airports it is necessary to use a combined measurement unit (as they used already internationally) and second (for realistic assessment and protection of the population) to have a maximum level for single noise events.
DISCUSSION

Q. (von Glarke) I agree with Dr. Jansen's statement that we have physiological responses to noise. We also heard in Dr. Covert's review about all of the physiological responses to noise which many of us think are very healthy and natural responses to our environment. Unlike we have the idea that one of these transient physiological responses becomes chronic or somehow lead to chronic diseases and pathological effects. It is really nothing to worry about. In all the research that I have followed over the past twenty years, I have not been able to come up with any clear-cut proof that there is a chronic health effect from the long-term noise exposure that we are talking about. I am not saying that these effects don't exist, but the only study which shows such a correlation was Jansen's study, which was cited before, on noise in industry. This was done fifteen or twenty years ago and hasn't been replicated since. This study showed a potential indication that noise exposure in industry might be correlated with some increase in cardiovascular disease. However, the same workers in noise that were studied had many other environmental factors associated with their work which might have been just as bad if not worse than the noise itself. Studies have been performed recently on mice and rats that show pathological effects to high-level noises but I think we should really concentrate on studies in the real-life situation that are made on man rather than on mice and rats. The stories we hear about malformations and reduced fertility in long-term studies of man and mice are open to some question. First, the noise levels are high. Second, it happens that there are not good controls used. When controls are handled in the same way the experimental animals are handled these effects disappear.

A. (Jansen) You mentioned my study of twenty years ago. Yes, it should be replicated and we are now undertaking experiments in order to find out the relevance of noise along with other factors in producing health effects on workers. We have a group of young men who are doing their research thesis just on this point. Perhaps it is possible within one or two years that we will have the results that you were asking about.

A. (Covert) Would like to answer Dr. von Glarke regarding the statement he just made. I hope that from the presentation I gave one didn't infer that there was an attempt to offer any clear-cut proof that there is a pathophysiological effect of noise, but rather that the indication is clear. It is clear in animals. Although we cannot necessarily apply animal studies to humans, nonetheless we do have the human studies of Dr. Jansen as well as studies done in Russia and Europe. Unfortunately, few studies have been done in the U.S. Part of my presentation was a plea for more activity in this problem area. I do feel, however, that prolonged exposure to noise, noise being unwanted sound, must act as a stressor. The effects of stress of accumulating stress have been present for over forty years. Most people now agree that stress is a factor which causes changes in pathophysiological problems. The stress of noise if noise does cause stress, and I think it does, can very well lead to pathophysiological effects. We should study this, whether we can ever say for certain that noise exposure for a given period of time at a given level is going to cause heart disease, it is possible that anomalies of certain kinds are not likely, but I think that certainly further studies need to be performed and that we agree on this point.

A. (von Glarke) I think it is simpler just to say that noise affects the quality of life and what we want to do is improve the quality of life.

A. (Ward) I disagree with Dr. von Glarke. Improving the quality of life is only one of the things we are after. More importantly, we are interested in the effects of noise on health. Noise is reported by the World Health Organization which includes such things as feelings of well-being. Let's talk about health as absence of pathology. True, we are interested in protecting the public's feeling of well-being in the long run but first let's concentrate on protecting the public from pathology.

A. (von Glarke) Noise is a stress, I agree. But we are exposed to many stresses during the day. Sitting on these chairs for eight hours is a stress and it just depends on how great the situation is. To all seriousness we once tried to follow some of Dr. Jansen's work and that of some other workers by obtaining vasoconstrictive responses to a vibration stimulus. We awakened the area of our subjects to vibration, we worked for hours, even days, until we had a nice response of vasoconstriction resulting from localized vibratory stimuli on the skin. We had this effect, finally, and suddenly the pointer went completely off the scale. It turned out that a young woman had walked through the room and our male subject was so stressed that the vaso-constriction from this stimulus was far more violent than from the laboratory vibratory stimulus.

Q. (Olson) How many people moved away from the Munich airport because the noise irritated them?

A. Those who moved did not do so because of noise nor were they found to be more sensitive to noise than was a control group.

Q. (Perdriel) I walked until all the papers were presented before asking any questions because I thought that one of the speakers might discuss the effect of noise on the visual system. For a number of years it is well known that exposure to high-intensity noise for several minutes or for several hours can bring about changes in the visual function and thus might endanger field safety. We have studied the effects of noise of 5,000 Hz, or a complex of 5,000 Hz, the intensity of which ranged between 05 and 105 db, on the parameters of the visual system and we have found a decrease of about 20% in the night vision capability during exposure to noise. This was determined by measuring the mesopic night threshold. These are objective values, secondly, in certain subjective values, particularly in certain subjects the time to perceive colored lights, as used in aviation, was greatly increased. Thirdly, the time needed to perceive ground relief features, or depth perception, was also greatly increased. It is
Important to find out the anatomical location responsible for these interactions. We believe that the thalamus may be the responsible site for interaction between the auditory and visual functions. In fact an efferent activity of the sensory visual and auditory processes (pathways) does take place in the thalamus. Based on electro-encephalographic studies of the thalamus it may be assumed that an interference (disorder) occurs at this site leading to a diminished passage of the sensory visual messages when noise stimuli traverse the thalamus. Moreover, an inverse study carried out in Italy confirms to a degree these findings. If one exposes the eye to a strong light for several minutes one finds a decrease in the auditory threshold. This proves again an interaction between the auditory and visual sensory messages.

A. There have been many experiments performed in these areas. We have done work in our own laboratory on these problems. I think that it is not justified to generalize from these experiments to the real life situation, for one must consider man has motivation and has capability to compensate and these parameters must be taken into account. What one needs to do is to make experiments under real life conditions rather than laboratory conditions. From the experimental situation we know many things, but it is very dangerous to generalize from experiments to the real life situation.
SECTION 11
### Summary for Studies on the Effects of Noise on the Cardiovascular System (CVS)

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anders Jonsson; Volvo Inc.; Gothenburg, Sweden. Lennart Hansson; Dept. of Medicine; Sahlgrens Hospital; 8-4345 Gothenburg, Sweden</td>
<td>1. Volvo Inc.; Gothenburg, Sweden</td>
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<table>
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<tr>
<th>Investigator's Phone No.</th>
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<tr>
<td></td>
<td>1. Volvo Inc.; Gothenburg, Sweden</td>
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<td></td>
<td>2. Bayer Farm; Sweden</td>
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<table>
<thead>
<tr>
<th># of Ref.'s</th>
<th># of Fig.'s</th>
<th>Language</th>
</tr>
</thead>
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<tr>
<td>9</td>
<td>2</td>
<td>English</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type &amp; duration of experiment</th>
<th>Purpose for study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Field survey - in auto factory</td>
<td>to test whether noise-induced irreversible hearing loss is associated with a permanent blood pressure (BP) increase.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Description of test groups (subjects)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>196 male automobile factory workers divided into three groups:</td>
<td></td>
</tr>
<tr>
<td>1. Tests - 44 with noise-induced hearing loss</td>
<td></td>
</tr>
<tr>
<td>2. Controls - 74 with normal hearing (same age)</td>
<td></td>
</tr>
<tr>
<td>3. 76-miscellaneous types of hearing damage (eliminated from survey)</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Control of other stresses</th>
<th>Statistical Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>No control - factory conditions</td>
<td>Students t-test - to compare BP in both groups; chi-square test to compare no. of hypertensives</td>
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</table>

<table>
<thead>
<tr>
<th>Noise Stimulus</th>
<th>CVS Response Measured</th>
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</thead>
<tbody>
<tr>
<td>Source: auto factory machine noise</td>
<td>Blood pressure (BP) measured after 15 min. test.</td>
</tr>
<tr>
<td>Spectral characteristics: not analyzed</td>
<td></td>
</tr>
<tr>
<td>Noise level: greater than 85 dB</td>
<td></td>
</tr>
<tr>
<td>Length of exposure: several years</td>
<td></td>
</tr>
<tr>
<td># of trials: not applicable</td>
<td></td>
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</table>

| Author's conclusions | Long exposure to stress, such as noise, may cause repeated increases in blood pressure that can lead to a permanent increase, due to a blood vessel adaptation. Noise-induced hearing loss was associated with hypertension in the auto workers, but no direct proof exists that the noise caused the blood pressure rise in this group. |

| Evaluation & comments | Sample groups too small - less than 100 per group. |

A survey of 196 male automobile factory workers was conducted to see if there was a permanent blood pressure increase in people exposed to noise levels high enough to induce permanent hearing damage. During routine physical exams, audiograms were performed in a sound-proof room. Out of the 196 subjects, 44 had a noise-induced hearing loss (greater than 65 dB at 3000, 4000, or 6000 Hz) and 74 subjects of the same age had normal hearing. Graphs of the loss of hearing in dB at each frequency level tested are included. The other 78 subjects had miscellaneous degrees of hearing loss and were excluded from the blood-pressure study. Blood pressures were measured once after 15 minutes rest in the 74 subjects with normal hearing and in the 44 subjects with severe hearing loss. The average systolic and diastolic blood pressures were higher (significant at P less than 0.0001 using students t-test) in the subjects with hearing loss than in those with normal hearing. There was also a greater proportion of hypertensive subjects (significant at P less than 0.05 using chi square test) in the hearing-impaired group. The results indicate that prolonged exposure to noise, which is strong enough to cause permanent hearing damage, may be a cause of a permanent blood pressure increase due to blood vessel adaptation to repeated stress.
variable showed a significant increase in the amount of acetone, and in the case of adhesive inhalation a significant response also for toluene (fig. 1b).

**DISCUSSION**

During the last few years there has been growing concern in the west of Scotland over the increasing number of young people who inhale solvent-based materials "for kicks". The results presented show that males predominantly engage in the practice. The age-group involved is 12 to 19 with a mean of 14·9 years. More than half (51·4%) were between 14 and 16 years of age. Although most (98·7%) cases were referred during the week these figures could be incomplete or even biased for two reasons. Firstly because "sniffing for kicks" is predominantly a group activity so that a clustering of cases in certain areas at certain times would almost certainly occur. Secondly, the source of referral in each case was the police who might well be fully occupied at weekends with other duties, etc. and therefore less likely to detect sniffers in a district. On the other hand the figures might represent a natural decline in snifflng practices at weekends, possibly because of the availability of other interesting and less deviant activities—e.g. football and youth clubs.

The observed clustering of cases which occurred during the morning, afternoon, and evening further supports our belief that this is, in the main, a group activity. At first glance, it seems as if the absence of referrals at periods during the day might coincide with police meal times. However, this was found not to be the case. We can only assume that the drop in reported episodes reflected the children's meal times, and that it represented not only an evening social activity but for almost half the cases an alternative to school attendance. It was not possible to determine from the information available whether truancy in these cases pre or post dated the snifflng history.

Although a wide variety of solvent-based materials in common domestic use are readily available to would-be sniffers, in practice 84% had chosen proprietary brands of adhesives. The method of inhalation showed even more conformity and undoubtedly reflected previous experience with solvents. It was interesting to note that the acute effects of these solvents most closely resembled alcoholic intoxication.

The analytical technique developed was rapid, reliable, and simple. Glues and other substances could be rapidly screened for suspected solvents thus confirming the formula stated by the manufacturer, although with respect to the wishes of certain manufacturers we have not listed the solvent formulations of the products investigated. The most popular glues involved were found to contain both acetone and toluene. The analysis of the blood-samples from sniffers readily detected these solvents.

Although this investigation has added to our current knowledge about the sniffers, the substances, the solvents, and the syndromes, it indicates a need for more factual information about the effects and toxicity of glues and other solvents available in household cleaning materials whose vapours could be inhaled "for kicks".

We thank Ann Magee for her technical assistance.

**REFERENCES**

Results for reprints should be addressed to J. S. O. Department of Forensic Medicine, The University, Glasgow G12 8QQ.

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**Occupational Health**

**PROLONGED EXPOSURE TO A STRESSFUL STIMULUS (NOISE) AS A CAUSE OF RAISED BLOOD-PRESSURE IN MEN**

**Anders Jonssén**

*Vasa Inc., Goteborg, Sweden*

**Lennart Hansson**

*Department of Medicine I, Sahlgrenska Hospital, Goteborg*

**Summary**

Systolic and diastolic blood-pressure was significantly higher in 24 male industrial workers with a noise-induced auditory impairment >85 dB at 2000, 4000 or 6000 Hz than in 74 males of the same age with normal hearing. Moreover, significantly more individuals with hypertension (resting diastolic blood-pressure >100/105 mm Hg) were found in the group with noise-induced loss of hearing. It is suggested that repeated and prolonged exposure to a stressful stimulus (industrial noise severe and prolonged enough to cause a permanent loss of hearing at the relevant frequencies) may be a contributing factor to the rise in blood-pressure through a mechanism involving structural adaptation of blood-vessels in response to repeated peaks of raised blood-pressure.

**INTRODUCTION**

Several kinds of mental stress are associated with a temporary rise in blood-pressure. In animal experiments it has been shown that repeated exposure to an altering stimulus can cause a permanent rise in arterial pressure. Such a relationship between repeated stressful stimuli and a permanent rise in blood-pressure is obviously not so easy to establish in man, partly due to the fact that environmental stress is difficult to measure and quantify.

The purpose of the present investigation was to determine whether a permanent rise in blood-pressure had occurred in individuals exposed to noise severe and prolonged enough to cause an irreversible loss of hearing.

**SUBJECTS AND METHODS**

The subjects were 196 male industrial workers. At a routinely performed health examination an audiometry test was done for each subject. The audiometry test was performed in a sound-proof room with a standard tone audiometer connected to earphones. Hearing test was set at the following frequencies: 250, 500, 1000, 2000, 4000, and 6000 Hz. The normal audiogram was defined as a loss of hearing less than 20 dB at all frequencies tested. A severe noise-induced impairment of hearing was defined as a loss of hearing amounting to 65 dB or more at 3000, 4000, or 6000 Hz (see accompanying figure). Individuals with an impairment of hearing <65 dB but >20 dB at these frequencies or a loss of hearing at other frequencies were excluded from the study.

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**REFERENCES**

Test frequency Hz

(A) A normal audiogram; (B) an audiogram showing a noise-induced auditory impairment with the characteristic "drop" at 4000 Hz.

Blood-pressure was measured in the recumbent position after the subject had rested for fifteen minutes. A mercury sphygmomanometer with a cuff containing a 10 × 10 cm rubber bag was used. Phase I (disappearance) of the Korotkoff sounds was taken as the diastolic blood-pressure. Blood-pressure was read to the nearest 5 or 10 mm Hg. The same nurse measured all the blood-presures and an audiometry assistant did all the audiometry tests. They were both unaware of the purpose of the investigation.

Upon completion of these examinations 74 subjects were found to have normal hearing whereas 44 had a severe noise-induced loss of hearing. The remainder, 78 subjects, had various forms of hearing impairment and constituted the "miscellaneous group" for which we did not further analyse.

Student's t test was used to compare systolic and diastolic blood-pressure in the two groups. The y2 test was used to compare the number of hypertensive individuals (defined as recumbent blood-pressure > 160/100 mm Hg after fifteen minutes of rest) in the two groups.

RESULTS

The average systolic and diastolic blood-pressure were significantly higher in subjects with a noise-induced loss of hearing than in subjects with normal hearing (see accompanying table). There were also significantly more hypertensive individuals in the group with impaired hearing than in the one with normal hearing (table).

DISCUSSION

There are three main findings in this study. First, out of 196 male industrial workers 44 had a severe loss of hearing at either 3000, 4000, or 6000 Hz but not at other frequencies indicating that this impairment was due to noise. Usually, prolonged exposure (several years) to severe noise (> 85 dB) is needed to cause damage of this severity. Secondly, resting blood-pressure was significantly higher (p < 0.05) in these men compared to 74 men of the same age but with normal hearing. Finally, the proportion of hypertensive subjects was significantly higher (p < 0.05) in the group with noise-induced loss of hearing as compared to the group with normal hearing.

Industrial noise can undoubtedly be regarded as a stressful stimulus, occasionally being powerful enough to cause pain. It is well known from animal studies that brief exposure to noise can cause a rise in blood-pressure. Exposure to noise has also been used as one of several means to raise blood-pressure acutely in normotensive and hypertensive subjects. However, little is known about prolonged exposure to noise and its effect upon blood-pressure in man. Noise exposure in these individuals had occurred during previous employment in, for example, shipyards or mechanical workshops. Obviously, it cannot be claimed with absolute certainty that exposure to noise caused the increased blood-pressure or the higher rate of "hypertension" observed in the group with noise-induced auditory impairment. On the other hand there were no other obvious differences between the two groups that could easily explain our findings.

Speculations that individuals with a genetic predisposition to develop hypertension are also more susceptible to noise or that such individuals seek out noisy or stressful jobs are not well founded and seem improbable. A more logical assumption is that repeated severe exposure to noise causes repeated rises in blood-pressure. This in turn could be expected to cause a permanent rise in blood-pressure in analogy with findings in animals repeatedly exposed to alerting stimuli. In addition individuals with a genetic predisposition to develop hypertension may "hyper-react" to stressful stimuli according to findings by Hallblidc who described a more pronounced rise of blood-pressure in young "prehypertensive" spontaneously hypertensive rats as compared to normotensive rats. Exposure to repeated peaks of blood-pressure could then be expected to cause a permanent rise in blood-pressure due to structural adaptation of the heart and resistance vessels.

We therefore feel that the most reasonable explanation to the presented findings is that prolonged exposure to a stressful stimulus may have caused repeated rises in blood-pressure leading to circulatory adaptations and a permanent rise in blood-pressure. We intend to follow up these preliminary findings with detailed examinations of larger numbers of individuals exposed to industrial noise.

We thank Dr. L. E. Peterson, Department of Statistics, University of Gothenburg, for the statistical analyses, and we gratefully acknowledge the skilled help of Mr. P. B. Ottosson and the support provided by Volvo Inc. and Hawa Forna of Sweden.

Requests for reprints should be addressed to L. H., Department of Medicine I, Sahlgren's Hospital, S-413 43 Gothenburg, Sweden.

REFERENCES

SECTION 12
<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
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<tbody>
<tr>
<td>Dr. L.P. Lipsitz (Prof. of Psychology and Medical Science, Brown Univ., Providence, R.I. 02912)</td>
<td>Brown University Providence, R.I.</td>
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<th>Investigator's Phone No.</th>
<th>Sponsoring Organization</th>
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<td>-</td>
<td>U.S.P.H.S. (Public Health Service); Grant Foundation</td>
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### | # of Ref.'s | # of Fig.'s | Language | English |
--- | --- | --- | --- | --- |
### | 1 | 1 | Purpose for study: To see if heart-rate response to noise can be used to identify neurological and behavioral problems in human infants

### Description of test groups (subjects, age, etc.):
- 16 essentially normal newborns divided into 2 groups:
  1. 4 males, 4 females—lesser number of non-optimal birth conditions (low-risk)
  2. 2 males, 4 females—greater number of non-optimal birth conditions (high-risk)

### Control of other stressors: drugs given to the mother pre-delivery were recorded; obstetrical difficulties during delivery were recorded.

### Noise stimulus
- Source: Hewlett-Packard Model 3300A audio oscillator connected to an electro-voice speaker
- Spectral characteristics: 700 Hz (habituation); 300 Hz (dishabituation)
- Noise level: background: 73 dB noise stimulus: 85 dB square wave
- Length of exposure: 5 sec. tone
- # of trials: 20 trials per subject; 5 sec. tone: 25 sec. outer

### Statistical Methods
- Mann-Whitney U-test; 2-tailed correlation t-test

### CVS Response Measured
- Heart rate (BPM)
- Heart rate acceleration and deceleration
- Non-auditory effects: no significant difference in heart rate response to noise over CVS; trials between groups; significant diff. was noted in averaged heart rate acceleration and deceleration; high-risk group: more heart rate acceleration; low-risk group: more, deceleration

### Author's Conclusion
Heart rate responses to noise may be useful in identifying infants with psychological problems, but no conclusive evidence is available. Obstetric history bears some relation to neurological problems, which might be detected using this cardiovascular response.

### Evaluation & Comments
This demonstrates some of the vegetative-type responses to acoustical stimuli in infants.

Heart rate responses to noise of normal infants with either high-risk or low-risk obstetrical histories were studied. High-risk infants had more adverse factors during their gestation and birth, such as abnormal intrauterine position, a cord abnormality, caesarean birth, or adverse maternal factors (such as drugs taken). The experiment was designed to see if the heart rate response to noise could be used to identify potential neurological and developmental problems. The subjects were 16 newborns, of which 8 were a low-risk group and 8 were a high-risk group. There were 4 males and 4 females in each group. The infants were tested while in a crib in a quiescent state inside a sound-attenuated chamber. The sound source was a Hewlett-Packard Model 3300A audio oscillator connected to an Electrovoice speaker. A background noise of 73 dB was present, due to the ventilation system. The noise stimuli included a habituation tone at 700 Hz and a dishabituation tone at 300 Hz. The sound level was 85 dB and the duration of the noise stimulus was a 5 second tone. The heart rates were monitored by EKG during the 18 minute experimental session for each infant. Each session consisted of 2 minutes quiet, a 10 minute period of 18 habituation trials and 2 dishabituation trials, and 2 minutes quiet. Each trial included a 5 second tone and then a 25 second interstimulus interval. Values were recorded for the highest and lowest heart rates in the pre- and post-stimulus periods. Acceleration scores were determined by subtracting the highest pre-stimulus heart rate from the highest post-stimulus heart rate. Deceleration scores were computed by subtracting the lowest post-stimulus heart rate from the lowest pre-stimulus heart rate. The expected response for the high-risk infants was a delayed habituation, indicated by an accelerated heart rate (which may be a defensive reaction to noise). Low-risk infants were expected to show the opposite effect of habituation to the noise (possibly an orienting reflex). Neither group of infants was definitely habituated to the noise. No significant difference in heart rate response over trials between groups was found. When averaged acceleration and deceleration scores were considered, high-risk infants showed significantly more acceleration and less deceleration than the low-risk group.
Obstetric History and the Heart-rate Response of Newborns to Sound

Steven Kittner Lewis P. Lipsitt

Introduction

It has been suggested that the inability to adequately evaluate the behavioral and neurological condition of a newborn infant is perhaps the greatest deficiency in perinatal medicine (Gluck 1974). Evaluative techniques are needed, not only by those concerned with the continuing welfare of the child, but also by the obstetrician and anesthesiologist who wish to know the consequences of their procedures.

Parmeece et al. (1974) developed a comprehensive approach to this problem, in which behavioral performances as well as pregnancy, perinatal, and neonatal biological events are included in the initial risk assessment of each newborn. The present study is an attempt to develop an additional behavioral measure for such a larger assessment battery.

The study was concerned with the relationship between obstetric history and newborn behavior. Its purpose was to determine whether two groups of infants differing in obstetric history would also differ in their heart-rate response to a series of auditory stimuli during the first few days of life.

The heart-rate response to auditory stimuli was selected as a test item for three reasons. First, the newborn response to auditory stimulation may be an important factor in the mother-infant interaction. It has been reported that newborns can respond to their mother's voice by head-turning as early as the third day of life and can distinguish this voice from other voices (André-Thumms and Ausgaarden 1966, Hammon 1970).

Secondly, studies employing other dependent measures have suggested that habituation may be delayed in newborns who have suffered perinatal complications (Bronzstein et al. 1958, Eisenberg et al. 1966). Schulman (1970) compared the heart-rate response to auditory stimuli of low-risk and high-risk pre-term infants and found a difference in the latency of the response. Although a significant decrement in the accelerative heart-rate response between trials 1 to 5 and 26 to 30 was found for both groups, Schulman did not use a novel auditory stimulus at the end of the procedure and hence it cannot be determined whether both groups habituated according to the definition of Thompson and Spencer (1966), which requires a test of dishabituation. The design of the present study allows one to check for differential habituation of the heart-rate response between two groups.

Thirdly, the direction of the heart-rate response is variable. D (1959) and five review response in and Cliffor tation is a o and is att contrast, g ested to b reflex ass Much wo interpreted (Graham a Clifton 19 there is ev heart-rate stabic ind period (Cl 1972. The present ex be increa decelerat a difficult those wil The nu condition was used each birth on the ba delivery a

Specific medication obtained, expected study of Aleksand know wh responsiv ante-part factors, variation.

If the stimulati disparate drug eff
response is also an important dependent variable. Drawing upon the work of Lacey (1959) and Sokolov (1963) and an extensive review of the research on heart-rate response in adult human subjects, Graham and Clifton (1964) suggested that deceleration is a component of the orienting reflex and is associated with stimulus intake. In contrast, cardiac acceleration was suggested to be a component of the defensive reflex associated with stimulus rejection. Much work with newborns has been interpreted in terms of these suppositions (Graham and Jackson 1970, Kearley 1973, Clifton 1974, Porges 1974). In addition, there is evidence that the direction of the heart-rate response to stimulation is a stable individual difference in the newborn period (Clifton and Graham 1968, Jacklin 1972). Thus another hypothesis of the present experiment was that there might be increased acceleration and decreased deceleration responses in infants who had a difficult birth history compared with those with a relatively benign history.

The number of non-optimal obstetric conditions from the list of Prechtl (1968) was used as a measure of the difficulty of each birth. Prechtl selected these variables on the basis of the risk of mortality during delivery and the first two weeks of life.

Specific information on the pre-delivery medication of the mother of each child was obtained, since drug conditions might be expected to have extraordinary effects in a study of this type (Bowes et al. 1970, Aleksandrowicz 1974). It is important to know whether an infant's psychobiological responsiveness is determined principally by ante-partum drugs, by multiple obstetric factors, or by individual genetic variations.

If the heart-rate response to auditory stimulation differentiates newborns having disparate birth histories, independently of drug effects, strong support would be given to the further study of this psychobiological measure as a newborn assessment form. Positive findings would also contribute towards an understanding of individual differences in newborn behavior.

Method
Sixteen newborns born at the Women and Infants' Hospital of Rhode Island were studied. The infants, all of them bottle-fed, were selected on the basis of the number of non-optimal obstetric conditions from Prechtl's list (Table I) entered in the infants' hospital records.

The following categories of infants were excluded: (1) infants of less than 38 weeks gestational age or weighing less than 2,495 g (5-1lb) at birth; (2) infants whose clinical condition at birth was such that they were placed in special-care nurseries; and (3) infants whose parents were living outside the Providence area, because of inconvenience for possible follow-up studies.

The first category prevented testing of infants who were pre-term, small for gestational age, or who had any known or suspected abnormality. Therefore the study was of essentially normal newborns, with greater and lesser recorded indications of 'perinarial hazard'. No infant was accepted for study without the written consent of the mother and the child's pediatrician.

The selected newborns comprised two groups: a 'low Prechtl' group (LP) with three or fewer non-optimal conditions, and a 'high Prechtl' group (HP) containing newborns with seven or more non-optimal conditions. There were eight infants in each group, four male and four female. Interestingly, the HP and LP groups had significantly different Apgar scores at 1 minute (t = 2.38, df = 14, p < .05), but this difference was reduced to nonsignificant effect at 5 minutes. Table II summarizes the characteristics of each group.
TABLE II

Mean and standard deviations of the low and high PCP groups

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<th>Characteristic</th>
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<th>High (n = 10)</th>
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<tr>
<td></td>
<td>M</td>
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<tr>
<td>Proactil score</td>
<td>3:15</td>
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<tr>
<td>Average gestation age (mm)</td>
<td>39:44</td>
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<tr>
<td>Estimated gestational age (wks)</td>
<td>35:40</td>
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<td>Birthweight (lbs)</td>
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<tr>
<td>Birthweight (lbs)</td>
<td>39:40</td>
<td>15:35</td>
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TABLE III

Maternal pre-delivery medication: drug and time weighting systems

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<tr>
<th>Medication</th>
<th>Drug code weighting system (d score: d = done)</th>
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<tr>
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<td>Drugs and dosage levels</td>
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<tr>
<td>Narcan</td>
<td>d = 50mg</td>
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<tr>
<td>Methadone</td>
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<tr>
<td>Secobarbital</td>
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<td>Penicillin</td>
<td>d = 100mg</td>
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<td>Prochlorperazine</td>
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<td>Promethazine</td>
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<td>Propranolol</td>
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<td>Promethazine-hydrochloride</td>
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</table>
Data Analysis
The values used for this experiment are the highest instantaneous rate (the shortest interval between beats) and the lowest instantaneous rate (the longest interval between beats) in the 10-second periods preceding and following the initiation of the auditory stimulus. These values will be referred to as prestimulus-high, prestimulus-low, poststimulus-high and poststimulus-low (Fig. 1).

From these data, acceleration and deceleration scores in beats per min (bpm) were computed for each stimulus trial for each infant. Acceleration scores were calculated by subtracting the highest heart-rate in the 10-second prestimulus period from the highest heart-rate in the 10-second poststimulus period. Deceleration scores were computed by subtracting the lowest heart-rate in the 10-second poststimulus period from the lowest heart-rate in the 10-second prestimulus period. It is important to note that the acceleration and deceleration scores were computed from two different sets of data, hence it would be possible for both a positive acceleration score and a positive deceleration score to occur on a single trial.

The data were uncorrected for initial level effects (Lacey and Lacey 1962, Wilder 1967, Graham and Jackson 1970, Stein- schnieder 1971) because there were no significant differences between the groups in pre-experimental basal heart-rate (two-minute sample), nor in their prestimulus-low and prestimulus-high values (averaged across the first 18 trials). Using the Mann-Whitney U test (Siegel 1956) with eight infants in each group, the U values were 31, 25 and 25 respectively.

Results
Figures 2a and 2b show the group trends of acceleration and deceleration scores across the 20 stimulus trials. (The acceleration and deceleration values for the two

Procedure
The infants were tested between 10.45 and 11.45 am, between the feedings which occurred at about 9.30 and 1.00. Each infant was brought from the nursery to the laboratory by a nurse, who attached EKG electrodes and the pneumobelt. The infant was then swaddled and placed on its left side to allow full view of the infant's face by the nurse and experimenter. Crying was the only behavioral state which excluded an infant from the experiment; for the most part the infants were in a quiescent state throughout but could have been awake or asleep, with eyes open or closed.

After calibration of the equipment there was a two-minute baseline recording period, a 10-minute auditory habituation procedure, and a second two-minute baseline period. The total time taken with each infant was approximately 18 minutes.

During the two baseline periods only heart-rate and respiration were recorded. The auditory habituation procedure consisted of 18 habituation trials and two dishabituation trials. Each trial consisted of a five-second tone followed by a 25-second interstimulus interval. The habituation tone was a 700Hz, 85dB square wave sound. The dishabituation tone was a 300Hz sound of the same dB level and waveform.
groups of infants are presented in the same figures for purposes of comparison.) It can be seen that in comparison to the LPR group, the HPR group shows consistently larger acceleration and smaller deceleration scores across the habituation trials.

In order to characterize these group differences further, each infant's acceleration and deceleration response was averaged across the first 18 trials. As expected, these group differences are highly significant (Table IV).

Table V shows the results of tests for a trials effect by comparing the average heart-rate acceleration and deceleration scores of trials 1 to 5 with those of trials 14 to 18 for each group. A two-tailed correlated t test showed no significant trend in acceleration scores for either group. However, a significant decrement in deceleration scores was found for both the LPR group (p < .001) and the HPR group (p < .02).

Because the HPR group increased in its acceleration response from the early to the later trials, while the LPR group decreased, the suggestion of such an interaction was examined statistically by calculating a difference in each infant's acceleration response from early to later trials. The two distributions of differences were then compared by a Mann-Whitney U-test to determine whether these distributions were different for the two groups: they were not.

Although there was a significant decrement across trials for the deceleration scores, there was little recovery of the deceleration response to the novel stimulus.
Fig. 1. Trends across trials of heart-rate acceleration and deceleration to auditory stimuli. (a) Left-hand graph shows average acceleration score in each block of two trials for Low Prechtl (LP) and High Prechtl (HP) groups. (b) Right-hand graph shows deceleration scores in the same manner.

Finally, the product moment correlations of heart-rate acceleration and deceleration scores with Prechtl score and other selected variables are shown in Table VI. Only the first two variables yielded significant correlations. The Prechtl score showed the strongest relationship to the deceleration scores, while maternal parity or the number of previous live births had the strongest relationship to the acceleration scores. It may be noted that the Appgar scores did not relate reliably to either acceleration or deceleration. This finding, coupled with the fact that the Appgar scores were reliably different only at 1 minute, suggests that the Appgar rating is not as effective in separating infants at risk as is the Prechtl classification scheme, at least in essentially normal, surviving babies.

Discussion

Heart-rate response trends over trials.
The HP group was expected to show a delayed habituation compared with the LP group. The results of this experiment

TABLE IV

<table>
<thead>
<tr>
<th>Heart-rate response (beats/min)*</th>
<th>Acceleration Prechtl group</th>
<th>Deceleration Prechtl group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>2.98</td>
<td>2.08</td>
</tr>
<tr>
<td>Mann-Whitney U test (two-tailed)</td>
<td>10 = 4, p = .01</td>
<td>11 = 5, p = .007</td>
</tr>
<tr>
<td>t Test (two-tailed)</td>
<td>t = 2.97, p &lt; .02</td>
<td>t = 4.12, p &lt; .01</td>
</tr>
</tbody>
</table>

*Averages across trials 1 to 18.

Although decrements in heart-rate recovery trials 19 and 3 on trials 19 and 20 (see Figure 2).

...
do not clearly support a finding of habituation of heart-rate responses in either group. Although Table V does show a significant decrement in the deceleration scores for both groups, there was no significant recovery of the deceleration response on trials 19 and 20 for either group; thus the minimal criteria of habituation (Thompson and Spencer 1966) have not been satisfied.

Direction of the heart-rate response. Although there were no significant differences between the two groups across trials, there were significant differences between the two groups for both acceleration and deceleration. The LER group had smaller acceleration and larger deceleration scores than the PRE group. The hypothesis that the PRE groups represent populations having differential reactivity to stimulation is supported: the group having a relatively easy birth history showed more signs of orienting to auditory stimulation during the first few days of life. Studies testing different modalities with different response measures such as a visual fixation (Sigman et al. 1973) would be valuable for corroborating the significance of these results.

Relationship of heart-rate response to obstetric variables. Only PREich score and maternal parity showed a significant relationship to acceleration or deceleration scores (see Table VI). That parity should have a comparable correlation to the total PREich score is not surprising, since all the CPR infants were born of primiparous mothers, while all but one LER subject were born of multiparous mothers. Being a

<table>
<thead>
<tr>
<th>Acceleration score</th>
<th>Trials 1-5</th>
<th>Trials 14-18</th>
<th>t correlated</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Preich group</td>
<td>-1.30</td>
<td>-0.50</td>
<td>1.34</td>
<td>N.S.</td>
</tr>
<tr>
<td>High Preich group</td>
<td>-1.10</td>
<td>-0.70</td>
<td>1.10</td>
<td>N.S.</td>
</tr>
<tr>
<td>Deceleration score</td>
<td>-1.00</td>
<td>-0.30</td>
<td>1.00</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Low Preich group</td>
<td>-1.50</td>
<td>-0.20</td>
<td>1.50</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>High Preich group</td>
<td>-1.40</td>
<td>-0.10</td>
<td>1.40</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

TABLE VI
Correlation of selected variables with heart-rate response to auditory stimuli for all infants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Heart-rate response scores</th>
<th>Acceleration</th>
<th>Deceleration</th>
</tr>
</thead>
<tbody>
<tr>
<td>PREich score</td>
<td>-0.80</td>
<td>-0.60**</td>
<td>-0.40</td>
</tr>
<tr>
<td>Maternal parity</td>
<td>-0.50*</td>
<td>-0.30</td>
<td>-0.10</td>
</tr>
<tr>
<td>Apnea score</td>
<td>-0.10</td>
<td>-0.30</td>
<td>-0.10</td>
</tr>
<tr>
<td>Age at testing</td>
<td>-0.20</td>
<td>-0.10</td>
<td>-0.10</td>
</tr>
<tr>
<td>Maternal pre-delivery medication drugs and barbiturates</td>
<td>-0.10</td>
<td>-0.05</td>
<td>-0.10</td>
</tr>
<tr>
<td>All drugs</td>
<td>-0.20</td>
<td>-0.10</td>
<td>-0.10</td>
</tr>
</tbody>
</table>

* p < .05, two-tailed test
** p < .01, two-tailed test
† Time between first pre-delivery medication and birth.
primiparous birth counts as a non-optimal obstetric condition on the Prechtl scale.

Parity has previously been suggested to co-vary both with the anxiety of the mother (Copans 1973) and with the use of general anesthesia for the mother (Moraes and Birch 1974). Friedman (1975) cites a study by Genzeli (1954) which reported that the level of 17-hydroxyprogesterone is higher in infants born of primiparous than of multiparous mothers. Paradoxically, Friedman (1975) found that infants born of high-parity mothers required significantly more trials on a visual habituation experiment than did infants born to low-parity mothers. This emphasizes the importance of using multiple response measures in any attempt to assess newborn behavior.

The maternal predelivery medication variables assessed (see Tables III and VI) showed no significant relationship with heart-rate acceleration or deceleration scores.

Conclusions

While heart-rate response trends over trials did not discriminate well between groups scoring high or low on Prechtl's scale of non-optimal maternal, parturition, and fetal conditions, significant differences between the 'high-risk' and 'low-risk' groups were found for the averaged heart-rate acceleration and deceleration scores. It is suggested that this difference may reflect a differential reactivity to stimulation in the two groups.

The reason for this study was that response measures which could differentiate infants with a low number from those with a high number of non-optimal obstetric conditions would be worth further study, since such measures could prove valuable as an additional test item in a larger assessment battery. While it is known that obstetric history bears some relation to the manifestation of neurological abnormalities, this study demonstrates that even in ostensibly normal infants, non-optimal obstetric factors are responsible for some measure for psychobiological functioning at birth. The question remains as to whether behavioral measures such as heart-rate response to stimulation can enhance our ability to identify infants in jeopardy for later developmental problems. This could be extremely important in the search for methods of prevention and of effective remedial treatment.

Acknowledgments: We are greatly indebted to Bernice Reilly, R.N., for her very special expertise in all facets of newborn research, and without whose conscientious dedication the data for this study could not have been obtained. The assistance of Dr. Charles Coccia, Dr. Patrick Burke, and Bonnie Zinger during various phases of this study is also gratefully acknowledged. We are indebted to Prof. Leo Stern, Chairman of the Section of Reproductive and Developmental Medicine, Brown University, for a critical reading of an earlier manuscript. Support for the study came from USPHS Grant No. HD 07911 and a research grant from the Great Foundation.

This study is a portion of an Honors Thesis conducted by Kinner (1973) under the direction of the second author.

AUTHORS' APPOINTMENTS

Lewis P. Lipsitt, Ph.D., Professor of Psychology and Medical Scientist: Director, Child Study Center, Brown University, Providence, Rhode Island 02912.

Dr. Steven Kinner is at present a student in the University of Pennsylvania Medical School, Philadelphia.

SUMMARY

Two groups of clinically normal newborns, differing in the number of non-optimal factors in their obstetric history, were compared by measuring heart-rate response to a series of auditory stimuli. There was a significant difference between the groups in the direction of the average heart-rate response. The 'high-risk' group showed greater heart-rate acceleration and less deceleration compared with the 'low-risk' group. The heart-rate responses were significantly related to the number of non-optimal obstetric conditions and...
to parity, but not to the maternal pre-delivery medication score. The greater the ‘risk’ at birth, the less was the deceleration; the greater the maternal parity, the less did acceleration occur in response to auditory stimulation. There was no significant difference between the high-risk and low-risk groups in heart-rate response trends over trials. Both groups showed reliable diminution of deceleration heart-rate response over trials.

Résumé

Histoire obstétricale et réponse de la fréquence cardiaque au son chez le nouveau-né

Deux groupes de nouveau-nés cliniquement normaux mais différents par le nombre de facteurs non optimaux dans leur histoire obstétricale ont été comparés par la mesure de la fréquence cardiaque à une série de stimuli auditifs. Il a été noté une différence significative entre les groupes dans le sens de la réponse cardiaque moyenne. Le groupe à ‘haut risque’ a montré plus d’accélérations et moins de décelerations par comparaison avec le groupe à ‘bas risque’. Les réponses cardiaques ont été significativement reliées au nombre de conditions obstétricales non optimales et à la parité, mais non au score de médication maternelle avant la naissance. Plus grand est le ‘risque’ à la naissance et moindre est la décelération; plus grande est la parité maternelle, plus faible est l’accélération en réponse aux stimulations auditives. Il n’y a pas eu de différence significative entre les groupes à haut et bas risque dans la réponse cardiaque au cours de la répétition des essais. Les deux groupes ont montré une diminution réelle de la décelération dans la réponse de fréquence cardiaque au cours des essais successifs.

Zusammenfassung

Schwangerschaftsannahme und das Ansprechen der Herzfrequenz auf Geräusche beim Neugeborenen


References


DEVELOPMENTAL MEDICINE AND CHILD NEUROLOGY. 1976, 18


Graham, P. K. (1964) "The ability of individual differences in heart rate activity during the newborn period." Psychophysiology, 2, 37.


Kron, B. L., Stein, M., Goldkind, R. E. (1966) "Newborn sucking behavior affected by obstetric solution." Pediatrics, 37, 1012.


Stedler, G. A. (1964) "Newborn attention as affected by medication during labor." Science, 144, 315.


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Monte, Cap.
SECTION 13
### Summary Form for

**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

| **Principal Investigator(s):** | Z. Koszarny  
| | G. Kozlowska  
| | W. Stefa |
| **Institution and address where research was performed:** | State Hygienic Institute of Warsaw  
| | Warsaw, Poland |
| **Investigator's Phone No.** | |
| **Sponsoring Organization** | |
| **Citation:** | Koszarny, Z. et al. The effect of airplane noise on the inhabitants of areas near the Okocie airport in Warsaw. Rozmiki Powszechnego Zakladu Higieny 27 (3): 113-121, 1976 |
| **θ of Ref.'s 15** | |
| **θ of Fig.'s 7** | |
| **Language** | Polish (partially translated) |

### Type & duration of experiment:

Survey type study  
(health questionnaire & noise disturbance scale)

### Purpose for study:

To record the types of health and psychological effects on people near a large airport in Warsaw.

### Description of test groups (subjects: θ = a, b, c, d, etc...):

- 511 people who lived in 2 areas near Okocie airport in Warsaw were surveyed:  
  - Area A: over 100 dB = 256 people  
  - Area B: 80-98 dB = 255 people  

  (divided into groups of men and women 20-70 yr old of similar social backgrounds)

### Control of other stressors:

No controls—field study

### Statistical Methods:

- Student t-test for annoyance test  
- Chi-square for health effects

### Noise stimulus:

- **Source:** Aircraft noise
- **Spectral characteristics:** Not given
- **Noise level:**  
  - Varies 1 area over 100 dBA  
  - Other area 80-90 dBA  
- **Length of exposure:** Varies
- **Number of trials:** Not applicable

### CVS response measured:

- **% who complained of frequent use of heart medication**
- **% who complained of cardiovascular problems**

### Nonauditory effects:

- Higher % of people in the greater noise area (A) complained of heart problems. This was statistically significant between the groups of women, but not with men.

### Other:

- Noise associated with greater annoyance, digestive problems, headaches, nervousness

### Author's conclusions:

There appears to be a relationship between aircraft noise and the general state of health of area residents. Significantly higher complaints were found in groups of women living in the area with over 100 dBA aircraft noise than women living in the 80-90 dBA area.

### Evaluation & Comments:

This article was only partially translated—sections of the article dealing with cardiovascular system and the experimental design are in English.

The psychological and physiological effects of aircraft noise were studied in residents of two areas near a large airport in Poland. A health questionnaire and a noise disturbance scale were used to survey 256 residents in area A (noise levels exceeding 100 dBA) and 255 residents in area B (noise levels of 80-90 dBA). The people surveyed were grouped according to sex, age, living conditions, education and socioeconomic level. The relative annoyance level for area A was 82% and for area B, 54%. The survey population had a normal distribution of psychological problems. The health questionnaires documented the frequency of complaints of various ailments. No statistically significant differences in complaints were found in groups of men living in the two areas. Significantly greater numbers of complaints related to the cardiovascular system, the digestive system, frequency of taking medication for heart problems or headaches, and nervousness were found in women living in the noisier area (A) than in women who lived in the lower noise level area (B). The results indicate that aircraft noise can have an effect on the general state of health. The authors note that many other stressful and adverse conditions besides noise may affect state of health, such as the work environments of the people studied.
EXPERIMENTAL METHODOLOGY AND MATERIALS

In our tests, we used a specific health questionnaire and a noise disturbance scale. The basics of working out the questionnaire and the method of collecting the material were discussed in an earlier article [8]. In the new tests, however, we made some changes in the way in which the disturbance scale was fashioned, so that it was possible to achieve better correlation between the replies and their order according to the importance of the question. In constructing the scale, we used the scale analysis method of Guttman [10], which is based on assigning each person being examined a degree of disturbance based on the level of his symptoms caused by airplane noise. We adopted a 5-degree evaluation scale. Any noise reaching a degree of at least 3 points, or 60%, was considered to be very harmful to the environment. This is the threshold above which systematic disorders in word perception occur. It was determined that airplane noise is the main cause of feelings of discomfort in persons found on the scale above this threshold.

A similar method of examining airplane noise disturbance has been used in England, France and the Benelux countries [2].

Our tests involved 256 residents of areas located in a region where the intensity level was higher than 100 dB (A) and 255 residents of an area with an intensity level of 80-90 dB (B). The tests were conducted during the winter months of 1974-75. The test groups were divided equally into men and women aged 20 to 70, of similar educational background, professional background, working conditions, but different social and residential conditions. Tables I, II and fig. 1 detail the professional and residential conditions of the populations of both regions examined.

The difference in the social and residential conditions could to a certain degree affect the validity of the results obtained in that the increased sensitivity to noise in persons having less favorable conditions of life; however, this is impossible to avoid, as it results from the very nature of the regions examined. In any case, this influence is not significant and will chiefly affect the residents of the areas with lower intensity levels [2,8].
### Table I
Age of Persons Examined

<table>
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<th>Sex</th>
<th>Avg. age of subjects in various age groups</th>
<th>Avg. age of subjects</th>
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<tr>
<td></td>
<td>20-36</td>
<td>37-53</td>
</tr>
<tr>
<td>Female</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>28</td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
<td>27</td>
</tr>
</tbody>
</table>

*A - region where 100 dB (A) 110
B - region where 80 dB (A) 90

### Table II
Living Conditions

<table>
<thead>
<tr>
<th>Evaluative factor</th>
<th>Indices</th>
<th>No. of persons or homes</th>
<th></th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td>Level of population</td>
<td>avg. no. of persons per room</td>
<td>1.39</td>
<td>1.31</td>
<td>0.08</td>
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<tr>
<td>Ownership</td>
<td>owner</td>
<td>30.9%</td>
<td>18.9%</td>
<td>3.16</td>
</tr>
<tr>
<td></td>
<td>renter</td>
<td>89.1%</td>
<td>81.1%</td>
<td></td>
</tr>
<tr>
<td>Outfittings</td>
<td>bathroom</td>
<td>37.3%</td>
<td>77.6%</td>
<td>11.23</td>
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<td></td>
<td>toilet</td>
<td>57.3%</td>
<td>85.8%</td>
<td>7.03</td>
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<td></td>
<td>running water and sewerage</td>
<td>69.9%</td>
<td>84.6%</td>
<td>4.02</td>
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<tr>
<td></td>
<td>central heating</td>
<td>28.9%</td>
<td>75.6%</td>
<td>11.95</td>
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<tr>
<td>Evaluation of living conditions</td>
<td>satisfied</td>
<td>30.5%</td>
<td>50.8%</td>
<td>4.77</td>
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<tr>
<td></td>
<td>not satisfied</td>
<td>69.5%</td>
<td>49.2%</td>
<td></td>
</tr>
</tbody>
</table>

*A - region where 100 dB (A) 110
B - region where 80 dB (A) 90
t - value of t-Student test
P - level of significance
n.i. - statistically insignificant
DISCUSSION OF RESULTS

The evaluation of acoustic conditions in the area of Okecie involved, among other things, determining noise sources and their degree of bothersomeness. The data are detailed in Table III, in which the individual positions were arranged according to the average degree of disturbance for both of the regions discussed.

Analysis of the material shows that airplane noise plays a fundamental and significant role among the various types of noise that occur in the areas. The number of persons who complained of this type of noise was 91.1% in the over 100 dB (A) area and 63.4% in the 80-90 dB (A) area. The percentage of people who consider the noise to be very disturbing is significant, at 54.5% and 24.4% respectively.

Street noise was the object of complaints from about 38% of the respondents, and noise from neighboring houses or residences also represents a certain problem, but rather only for residents of areas with a lower airplane noise intensity level. It should also be pointed out that the number of people who indicated street and neighboring residence noise as very disturbing is small (not over 13%). The remaining types of noise sources are not a disturbance to the residents of the areas discussed.

In light of the differences in bothersomeness between the various types of noise occurring in both areas examined, only airplane noise
can, practically speaking, be taken as a basis for further analyses and comparisons. This concerns the goals of the tests, and above all determining the actual bothersomeness of airplane noise and its connection with physical parameters and the effect of the noise on the residents' health.

Table III
Evaluation of the acoustic climate in the area of Okcie

<table>
<thead>
<tr>
<th>Location</th>
<th>A. Sound intensity</th>
<th>B. Sound acoustic</th>
<th>C. Acousticity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>C</td>
</tr>
<tr>
<td>Czaplinia</td>
<td>4.5</td>
<td>5.5</td>
<td>5.1</td>
</tr>
<tr>
<td>Rusz 1</td>
<td>3.1</td>
<td>3.0</td>
<td>3.3</td>
</tr>
<tr>
<td>Zakłady próby samolotów</td>
<td>4.5</td>
<td>4.5</td>
<td>4.5</td>
</tr>
<tr>
<td>Zakłady dozynia</td>
<td>5.1</td>
<td>5.1</td>
<td>5.1</td>
</tr>
<tr>
<td>Pralnica muszkiety</td>
<td>6.1</td>
<td>5.5</td>
<td>5.5</td>
</tr>
<tr>
<td>Rusz 2</td>
<td>7.0</td>
<td>7.0</td>
<td>7.0</td>
</tr>
<tr>
<td>Rusz loniey</td>
<td>54.5</td>
<td>54.5</td>
<td>54.5</td>
</tr>
</tbody>
</table>

A - region where 100dB (A)<110
B - region where 80dB (A)<90
P - level of significance
n.i. - statistically insignificant

Key:
1. noise source
2. number of subjects (in%) who indicated noise as:
   a. very disturbing
   b. somewhat disturbing
   c. not disturbing
3. Household devices
4. Railroad noise
5. Factories
6. Service establishments
7. Neighboring residences
8. Street noise
9. Air traffic noise

The measuring scale used made it possible to determine the average degree of disturbance of air traffic and airplane noise, which is 4.1 points for the area with the highest intensity level, and 2.8 for the other. Expressing the above figures in relative values, we can say that the disturbance of airplane noise in the first area is 32%, and 54% in the second (fig. 2).
The disturbance is generally worst during daylight hours, as
night flights at the airport near Okcie are sporadic.

Based on the scale measurements and the comparative examinations
(2), air traffic noise in the area of more than 100 dB (A) can be
considered very disturbing, while the noise in the area of 80-90 dB
(A) can be considered moderately disturbing noise, approaching the
upper limit. However, it is highly probably that as the intensity
of the air traffic increases in a given region, its percentage of
disturbance will also rise. This increase, as the examinations showed,
is approximately proportional to the logarithm of the frequency with
which airplanes pass by [2, 12].

The examinations conducted also showed a close correlation between
evaluation of the noise disturbance and the objective measure of its
intensity. This connection is very strong, if we take into considera-
tion the average noise disturbance of the region in question (the
correlation coefficient is 0.86), but is insignificant if we consider
the individual sensitivity of the persons examined (the correlation
coefficient is 0.42). Thus, the level of noise intensity alone does
not determine its degree of disturbance. Individual personality
factors, social and living conditions, type of work done and even
attitudes toward flying also affect the way in which one feels the
disturbance of noise [8, 13]. Alexander [2] indicates that these
factors can affect a change in the disturbance only to a certain degree.
However, their effect is insignificant if the noise disrupts everyday
activities.
Agreement between the evaluation of the noise disturbance level and the objective measure of its intensity indicates that personal bases for determining the bothersomeness of air traffic noise should be adopted. However, the fact that in areas recognized, on the basis of noise intensity levels, as being suitable for residence and school construction, up to 65% of the persons examined gave negative evaluations of the acoustic conditions is rather disturbing.

The percentage index of disturbance is also high, and though it is within the limits of so-called moderate noise, it indicates significant deterioration in the comforts of residence in the areas discussed.

Evaluations of the state of health and general well-being of the populations in the areas discussed were made on the basis of the frequency and intensity of symptoms of general ill-feeling and a poor state of health in groups of persons residing under varying acoustic conditions.

The following symptoms were observed:

1. In the overall evaluation of the state of health no real differences were noticed between the men of both areas examined ($\chi^2$ square = 0.11), but there were marked differences among the women ($\chi^2$ square = 4.114, $p = 0.04$).

2. No statistically significant differences in tests for neuroticism and extraversion were noted in either the men or the women. The distribution of the results relation to these characteristics is comparable to the distribution observed in the overall Polish population.

3. In analyzing the frequency of occurrence of specific symptoms of ill-feeling and a poor state of health, only in women living under worse acoustic conditions did we find a more frequent occurrence of general complaints related to the heart and the digestive tract, more marked feelings of fear and ill-ease, more frequent nervousness, and more numerous cases of taking medication for headaches or cardiac troubles.

4. Independent of the differences found between the areas discussed, frequent appearance among the subjects of complaints of chronic fatigue, troubled vision, relentless headaches, nervousness, trouble in hearing and frequent use of headache or cardiac medication should be emphasized. Numerical data are given in tables IV and V.
Based on these results, it is difficult to draw any final conclusions regarding the effect of acoustic conditions on the state of health of residents in areas near airports. The high percentage of people who complain of feelings of fatigue, headaches, trouble in hearing, frequent nervousness, cardiovascular troubles and digestive tract disorders, and the differences in the frequency with which symptoms of illness and general ill-feeling occur in women living under poorer acoustic conditions seem to testify to a relationship between air traffic noise and the state of health. On the other hand, we cannot exclude the possibility that the above indices are the result of other causes related to unsatisfactory environmental conditions, especially the working environment.

The observed differences in the effect of acoustic conditions in one's place of residence on the state of health of men as opposed to women seem to indicate that such an interpretation should indeed be adopted. For among women there was found a more numerous group of persons either not working (23% more than the men), or working under better acoustic conditions (13% compared to the men). The varying acoustic conditions in the place of residence could thus play a more decisive role in determining women's state of health than they do in men's, among whom acoustically unfavorable working conditions prevailed (60%). Specific and detailed explanation of this puzzle would require additional examinations and tests.

Table IV
Frequency of occurrence of symptoms of ill-feeling and illness under varying acoustic conditions

<table>
<thead>
<tr>
<th>Health indices</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A,%</td>
<td>B,%</td>
</tr>
<tr>
<td>Insomnia</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>Fatigue</td>
<td>71</td>
<td>65</td>
</tr>
<tr>
<td>Headaches</td>
<td>23</td>
<td>23</td>
</tr>
<tr>
<td>Fear or ill-ease</td>
<td>17</td>
<td>11</td>
</tr>
<tr>
<td>Nervousness</td>
<td>34</td>
<td>39</td>
</tr>
<tr>
<td>Taking headache medication</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td>Taking sleeping pills</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Taking heart medication</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td>Troubled vision</td>
<td>35</td>
<td>48</td>
</tr>
<tr>
<td>Trouble in hearing</td>
<td>34</td>
<td>40</td>
</tr>
<tr>
<td>Digestive troubles</td>
<td>22</td>
<td>19</td>
</tr>
<tr>
<td>Cardiovascular troubles</td>
<td>24</td>
<td>20</td>
</tr>
</tbody>
</table>

A = 100 dB < A < 110
B = 80 dB < A < 90
Table V
Relation between objective evaluation of air traffic noise and the
selected indices of state of health

<table>
<thead>
<tr>
<th>Health indices</th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>X^2</td>
<td>P</td>
<td>X^2</td>
<td>P</td>
</tr>
<tr>
<td>Sleep</td>
<td>0.527</td>
<td>n.i.</td>
<td>0.157</td>
<td>n.i.</td>
</tr>
<tr>
<td>Fatigue</td>
<td>0.040</td>
<td>n.i.</td>
<td>0.343</td>
<td>n.i.</td>
</tr>
<tr>
<td>Headaches</td>
<td>0.081</td>
<td>n.i.</td>
<td>0.691</td>
<td>n.i.</td>
</tr>
<tr>
<td>Fear or ill-ease</td>
<td>2.015</td>
<td>n.i.</td>
<td>3.843</td>
<td>0.05</td>
</tr>
<tr>
<td>Nervousness</td>
<td>0.031</td>
<td>n.i.</td>
<td>5.161</td>
<td>0.03</td>
</tr>
<tr>
<td>Taking headache medication</td>
<td>1.734</td>
<td>n.i.</td>
<td>6.871</td>
<td>0.01</td>
</tr>
<tr>
<td>Taking sleeping pills</td>
<td>1.025</td>
<td>n.i.</td>
<td>3.641</td>
<td>n.i.</td>
</tr>
<tr>
<td>Taking heart medication</td>
<td>0.398</td>
<td>n.i.</td>
<td>3.830</td>
<td>0.05</td>
</tr>
<tr>
<td>Troubled vision</td>
<td>0.110</td>
<td>n.i.</td>
<td>1.090</td>
<td>n.i.</td>
</tr>
<tr>
<td>Troubled hearing</td>
<td>1.012</td>
<td>n.i.</td>
<td>0.542</td>
<td>n.i.</td>
</tr>
<tr>
<td>Digestive troubles</td>
<td>0.301</td>
<td>n.i.</td>
<td>3.843</td>
<td>0.05</td>
</tr>
<tr>
<td>&quot;Heart&quot; troubles</td>
<td>0.398</td>
<td>n.i.</td>
<td>4.416</td>
<td>0.04</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>0.821</td>
<td>n.i.</td>
<td>0.046</td>
<td>n.i.</td>
</tr>
</tbody>
</table>

X^2 - value of chi square meter
P - level of significance
n.i. - statistically insignificant
Wpływ hałasu samolotowego na mieszkańców regionów przylotniskowych lotniska Okęcie w Warszawie

Zakładu Higieny Komunalnej Powszechnej Stała Wiedza.

Wstęp

Zagadnienie wpływu hałasu ścisłego na mieszkańców obszarów przylotniskowych jest przedmiotem szczególnego zaинтересowania licznych ośrodków naukowych.

Główny kierunek badań skupia się na ustaleniu rozmiarów i stopni zagrożenia zdrowia ludności oraz określeniu uczuleniowości hałasu lotniczego [1, 2, 5, 7, 8]. Wielu autorów zajmujących się powyższym problemem stwierdza u mieszkańców regionów przylotniskowych nasilenie takich objawów jak bóle głowy, brak spodu, trudności w koncentracji uwagi, zespół w zasypianiu, częste zakłócania snu, uczucie zmęczenia oraz zaburzenia czynności układów sercowo-naczyniowego i oddechowego, ubytki wLLLLLLL

Innym niemalże istotnym zagadnieniem wpływu hałasu samolotowego jest próba znalezienia możliwości właściwego powiązania między fizycznymi parametrami okoliczności a jego uczuleniowości dla mieszkańców [3, 9, 11, 12]. Dowożenie wyniki badań wskazują na możliwość wykorzystania stałej uczuleniowości hałasu do opracowania bardziej dokładnych metod oceny hałasu lotniczego.

W badaniach oddziaływania hałasu lotniczego w rejonie Okęcia staram się uwzględnić oba wspomniane wyżej kierunki badań. Główny na

W badaniach oddziaływania hałasu lotniczego w rejonie Okęcia staram się uwzględnić oba wspomniane wyżej kierunki badań. Główny navigator podarował na ocenę stanu zdrowia mieszkańców i ustalenie uczuleniowości hałasu w różnych strefach nasilenia. Podjęto również próbę oceny czynności stanowionych metod pomiaru hałasu lotniczego z subiektywną oceną jego uczuleniowości.
METODYKA BADAN I MATERIAŁ BADAWCZY


Podobną metodę badan uciążliwości halasu lotniczego zastosowała w Anglii, Francji i w krajach Beneluksu [3].

W ramach niniejszej pracy badaniami objęto 364 mieszkańców terenów położo-nych w strefe w podziale na strefę powyżej 100 dB (A) oraz 125 mieszkańców rejonu w podziale na strefę pomiędzy 100 a 105 dB (A). Badań przeprowadzono w miastach zimowych w dniach 20-25 stycznia 1973 r. Grupę badanych osób reprezentowały się w równym stanie przez mężczyzn, a przez kobietę w wieku 20-70 lat, o podobnym typie wzmocnienia, zawodzie, warunkach pracy, ale różnicujących wieku i środowiskowego-bytowych. Tabelę I, II i III charakteryzują różne formy zaników, a skale mięśniowo-ludzkich ludzi bez pracujących rejonów.

Oznaczenie warunków mieszkaniowo-bytowych może w pewnym stopniu wpły-wać na ocenę warunków ulegającychInfrastructure. Przypomnimy, że poprawia się wynik, a jego warunki w „wczesnym” chrzestnym. Wskaźnik tej jednak jest niewielki i dotyczą głównie mieszkańców rejonów o niższych strefach nasilenia [2, 3].

<table>
<thead>
<tr>
<th>Tablica I</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Wiek ludności</th>
<th>20-25</th>
<th>26-35</th>
<th>36-45</th>
<th>46-55</th>
<th>56-65</th>
<th>66-75</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kobiety</td>
<td>28</td>
<td>25</td>
<td>44</td>
<td>45</td>
<td>43</td>
<td>41</td>
</tr>
<tr>
<td>Mężczyźni</td>
<td>29</td>
<td>27</td>
<td>45</td>
<td>44</td>
<td>42</td>
<td>40</td>
</tr>
</tbody>
</table>

A — wyn. 100% (A)c=100
B — wyn. 100% (A)c=50

OMOWIENIE WYNIKÓW BADAN

Ocena warunków akustycznych w rejonie Okrąg odbyta między innymi określeń zieleni halasców i stopni ich dokształcenia. Ze względu na powyższe wstępne oceny uciążliwości dla obu omawianych rejonów.
Tab. 11
Warunki mieszkaniowe

<table>
<thead>
<tr>
<th>Napajalowy</th>
<th>Napajal</th>
<th>tak</th>
<th>nie</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1,38</td>
<td>1,11</td>
<td></td>
<td>0,68</td>
</tr>
<tr>
<td>B</td>
<td>1,38</td>
<td>1,11</td>
<td></td>
<td>0,68</td>
</tr>
</tbody>
</table>

A — rejon 10-15 mm (A), B — rejon 16-25 mm (B), P — rejon 26-35 mm (P)

A — wieczór, B — wieczór

Z analizy materiału wynika, że spośród bałwów występujących na danym terenie gatunkową rolę odgrywa bałtka leśnica. Liczba osób skarżących się na tego rodzaju hałas wynosi w strefie powyżej 100 dB (A) — 31,1% mieszkańców, natomiast w strefie o nachyleniu 85-90 dB (A) — 63,1%. Udział osób oczekujących bałtak jako bardzo niesprawny jest znaczyi i wynosi odpowiednio 54,3% i 24,9%.

Hałas uliczny stanowi przedmiot skąpania około 82% osób respondentów, a hałas dochodzący z sąsiednich mieszkań przedstawia pewien problem, szczególnie dla mieszkańców rejonu o mniejszym poziomie naczenia hałasu leśniczego. Należy przy tym zaznaczyć, że liczbę osób oczekujących hałas uliczny i z sąsiednich mieszkań jako bardzo niesprawny jest niedoceniony.
<table>
<thead>
<tr>
<th>Źródło bagnia</th>
<th>Letnie wiosenne (25^\circ)</th>
<th>Letnie wiosenne (30^\circ)</th>
<th>Letnie wiosenne (35^\circ)</th>
<th>Letnie wiosenne (40^\circ)</th>
<th>Letnie wiosenne (45^\circ)</th>
<th>Letnie wiosenne (50^\circ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>0.8</td>
<td>0.1</td>
<td>0.07</td>
<td>0.07</td>
<td>0.07</td>
<td>0.07</td>
</tr>
<tr>
<td>B</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>C</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>D</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>E</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

A — osada 100-400 [m] (d)</d>
B — osada 400-700 [m] (d)
C — wysokość [m] L-Studnia
D — poziom bezwodni
E — niewierzy typu stałej

A = osada 100-400 [m] (d) < 400
B = osada 400-700 [m] (d) < 700
C = wysokość [m] L-Studnia
D = poziom bezwodni
E = niewierzy typu stałej
(nie przekracza 12%). Pomiatale rodzaje źródeł hałasu nie stanowią docenialności dla mieszkańców omawianego rejonu.

Ponieważ więc stwierdzonych różnic w dokuczliwości pomiarów różnych rodzajów hałasów występujących w obu badanych strefach jedynie hałas samolotowy może stanowić, praktycznie rzecz biorąc, podstawę do dalszych analiz i porównań. Dotyczy to zwłaszcza istotnych cech badań o znaczeniu określających uciążliwości hałasu lotniczego i jej powiązań z parametrami fizjologicznymi oraz wpływem hałasu na zdrowie mieszkańców.

Zastosowana średnia pomiarowa powodowała na ustalenie przeciętnego stopnia uciążliwości hałasu lotniczego, który wynosi dla strefy o najwyższych poziomach nałączenia 4,1 punktów, dla drugiej natomiast 2,8. Wrażają one mnie o stosunkowi w wartościach względnych można powiedzieć, że uciążliwość hałasu samolotowego w pierwszej z omawianych stref wynosi 82%, w drugiej natomiast 34% (ryc. 2).

Wspomniana uciążliwość odnosi się zarówno do godzin dziennych, pomiarów nocy nocnych związanym z lotniskiem Orężel występują tylko sporadycznie.

![Diagram](image)

Ryc. 1. Uciążliwość hałasu lotniczego

Zgodnie z założeniami skali i badaniami porównawczymi (2) należy uznać hałas lotniczy w strefie powyżej 100 dB (A) za bardzo uciążliwy, natomiast hałas w strefie 80—90 dB (A) za hałas o umiarkowanej dokuczliwości, zblizający się do jej górnej granicy. Jest jednak wysoce prawdopodobne, że przy wzroście intensywności ruchu lotniczego w danym rejonie wzrośnie również wartość procentowa jego uciążliwości. W ten sposób, jak wykazały badania, jest w przybliżeniu proporcjonalny do negatywnych czynników zastosowaniu samolotów (2, 12).

Przyznają się budzące wykazywać ponadu występowanie cechowej korelacji między nacnym uciążliwością hałasu a obiektywnym pomiarem jego natężenia. Znajduje ten jest bardzo silny jeżeli biorze się pod uwagę średnią uciążliwość hałasu bariery rejonu (współczynnik korelacji wynosi 0,65), jest natomiast niewielki, jeżeli uwzględnia się indywidualną wrażliwość osób badanych (współczynnik korelacji wynosi 0,05). Tak więc tam panuje niewielka uciążliwość

Nr 2

Hałas samolotowy z zdrowia mieszkańców

Zgodność między oceną uciążliwości haluszu z obiektywnym pomiarem jego natężenia wykazuje na przyjęcie właściwych zasad określania uciążliwości haluszu, które mogą jednak ułatwić, że na terenach wzmocnionych pod względem intensywności haluszu zaatropenia budzące się do zamieszkania i szkolenia u wszystkich badanych występują negatywne odczucia akustyczne.

Procentowy wskaźnik uciążliwości również jest wysoki i chociaż może się w granicach tzw. haluszu umiarkowanego, wskazuje na znaczną pogorszenie komfortu zamieszkania w omawianym rejonie.

Oceny stanu zdrowia i zamieszczająca ludności badanych rejonów dokonano na podstawie częstotliwości występowania i nasilenia objawów zlego samopoczucia i zlego stanu zdrowia w grupach osób zamieszkałych w różnych warunkach akustycznych.

Zauważono następujące zjawiska:

1. Przy kompleksowej ocenie stanu zdrowia nie stwierdzono istotnych różnic między mężczyznami obu badanych rejonów (chi kwadrat = 0,11), występowały natomiast istotne różnice wśród badanych kobiet (chi kwadrat = 4,11; p = 0,04).

2. Zarówno wśród mężczyzn jak i wśród kobiet nie stwierdzono znaczących statystycznie różnic w badaniach neurologicznych i słuchowych.

3. Analizując częstotliwość występowania poszczególnych objawów zlego samopoczucia i zlego stanu zdrowia stwierdzono, że wobec haluszu zlego samopoczucia w grupach osób zamieszkałych w dorzeczu rzeki występowanie ogólnych objawów zlego samopoczucia oraz objawów lokalnych, a szczególnie objawów, które należy oceniać jako objawy nierealizowane.

4. Niezależnie od różnic jakie wystąpiły między omawianym rejonem należy podkreślić częste pojawianie się wśród badanych osób narzekających na chroniczne zmęczenie, zakażenie zębów, upośledzenie horyzontu, utratę słuchu oraz częste zakażanie języka oraz błon głowę i niemowlęcia, szczególnie dzieci.

Na podstawie przedstawionych wyników badań trudno jest wyleczyć ostateczne wnioski na temat wpływu warunków akustycznych na stan zdrowia mieszkańców rejonów przyłotniskowych. Wiele procent osób starających się na zdrowie, mały powrót, upośledzenie horyzontu, częste zakażowanie języka oraz błon błon głowę i niemowlęcia, szczególnie dzieci. Wśród nich również w części(239,775),(759,990)
| Nr 2 | Hale samotne z zdrowia mieszkańców |

**Tabela IV**

Często występowanie objęć nego odniesienia i objęć chorych z wybranych wykazanych

<table>
<thead>
<tr>
<th>Wskaźniki stanu zdrowia</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zakrzewienie amon</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Zakrzewienie zanieczyszczenie</td>
<td>15</td>
<td>22</td>
</tr>
<tr>
<td>Bile głowy</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Czerwone objęcie boku niepłukane</td>
<td>50</td>
<td>40</td>
</tr>
<tr>
<td>Krzewonie</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Przyjaznienie leków od boku głowy</td>
<td>15</td>
<td>22</td>
</tr>
<tr>
<td>Przyjaznienie leków zanieczyszczone</td>
<td>15</td>
<td>22</td>
</tr>
<tr>
<td>Zakrzewienie zanieczyszczenie</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Upośledzenie słuchu</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Dobroczynność zdrowia</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Dobroczynność zanieczyszczenie</td>
<td>20</td>
<td>20</td>
</tr>
</tbody>
</table>

A — 100-400 (A100)
B — 400-900 (A1000)

**Tabela V**

Zakrzewienie ubiegłych objętych w tym hale odniesienia z wybranymi wskaźnikami stanu zdrowia

<table>
<thead>
<tr>
<th>Wskaźniki stanu zdrowia</th>
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<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zakrzewienie amon</td>
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<td>n.d.</td>
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<tr>
<td>Zakrzewienie zanieczyszczenie</td>
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</tr>
<tr>
<td>Bile głowy</td>
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<td>n.d.</td>
</tr>
<tr>
<td>Czerwone objęcie boku niepłukane</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Krzewonie</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Przyjaznienie leków od boku głowy</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Przyjaznienie leków zanieczyszczone</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Zakrzewienie zanieczyszczenie</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Upośledzenie słuchu</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Dobroczynność zdrowia</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Dobroczynność zanieczyszczenie</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
<tr>
<td>Nefroczynność</td>
<td>0,000</td>
<td>n.d.</td>
</tr>
</tbody>
</table>

n.s. — wartość nieznaczna
n.d. — wartość nieobliczana

z tabela uwagi objętych w tym hale odniesienia z wybranymi wskaźnikami stanu zdrowia.
Zobawiany w różnie wpływ warunków akustycznych w miejscu zamieszkania na stan zdrowia mężczyzn i kobiet zaobawia się przemawiać za przyjęciem takiej właśnie interpretacji. Wśród kobiet bowiem znając się liczba grupa osób była to niepracy, a wnioski zawodowe (o 12% więcej anżyli u kobiet), bądź też przeciwnik w lepszych warunkach akustycznych (o 13% w stosunku do mężczyzn). Obserwacja warunków akustykowych w miejscu zamieszkania mogły więc odgrywać bardziej dożylną rolę w zarządzaniu stanu zdrowia kobiet anżyli u mężczyzn, u których przeważają prawdopodobnie cierpienia są pod względem akustykowym warunki pracy (60%). Szczegółowe wyjaśnienie tego zaszczytnia wymaga dalszych badań.

**WYNIKI**

1. Hala lotnicza o poziomie ponad 100 dB (A) stanowi znaczne obciążenie dla mieszkańców. Hala o tym poziomie należy uznać za bardzo nieudolny a tym niemniej się do zabudowy miasta.

2. Hala lotnicza o poziomie 80—90 dB (A) przy dochodzącym ruchu lotniczym stanowi umiarkowaną docenę dla mieszkańców. Należy jednak zawsze z możliwością podnielenia jego użyciowości w miarę wzrostu ruchu lotniczego. Jest to tym bardziej prawdopodobne, że wczoraj ujawniony w danych rejonach jest już obecnie bliski wartości granicznej.

3. Załączność między niektórymi obowiązującymi zgodnością warunkami akustycznymi w miejscu zamieszkania i objawami chorobowymi aby stan zdrowia mieszkańców wskazuje na prawdopodobieństwo ujemnego wpływu hali lotniczego na stan zdrowia mieszkańców.

**PIESEŃNICTWO**

THE EFFECT OF AIRPLANE NOISE ON THE INHABITANTS OF AREAS NEAR THE LOTOS AIRPORT IN WARSAW.

Summary

Investigations were carried out on 511 inhabitants of areas near the airport who were exposed to various characteristics of noise. Very high annoyance effects of airplane noise of intensities over 105 dB (A) were demonstrated and a moderate annoyance effect of noises of intensities below 85-90 dB (A).

A high proportion of subjects complained about a feeling of fatigue, headaches, hearing impairment, cardiac and gastrointestinal symptoms. The association of certain conditions with irritation, complaint of marine and olfactory symptoms was demonstrated only in women.

1. Praca należy udziwić o wykorzystanie efektównego aparatu. W konsekwencji nie należy wychudzać zwów zamazanymi literami lub czynków mierzonej czynności to ujawni się odbiornik przez podkreślenie słów czynnych i nieczynnych. Wszystko inne podkreślać a nieciekawe.

2. Objętość pracy nie powinna przekraczać w czasie 12 stron maszynopisu, zawsze jednak wyraźnie, łącznie z ilustracjami, tabelami, rysunkami i rysunkami.

3. Praca wstępna lub opracowana wg zasad pierwszego piątego i piątego w formie bezprzewodowej.

Przy udzielenia nie przyjęł powyższych stanowisk, należy je objaśnić w miejscu, gdzie pozwala na to ich przyjęcie. W przypadkach teoretycznych na podstawie tekstu pracy Redakcja nie udziela wartości i nie przyjmuje do poznawania w rozpuszczalniki. Poprawiona i przepisana praca należy przełożyć na papier w 2 egzemplarzach w ciągu 10 dni. Jeżeli praca nie została przyjęta do druku, informacja ozwarcie i czerwone tło, na którym napisane jest "Kościół Przyjaciół".

6. Na pierwszej stronie pracy, na wysokości 2/4 strony (wijn małe od kibła, potrójne jest na uwagę, techniczno-wykonawcze układy wymienić pełne imię i nazwisko autora lub autorów, tytuł pracy, nazwę zakładki i numer pracy pochodzi, tytuł naukowy, nazwisko i nazwisko i serwissu należącego zakładki, portfel pracy, 3-budowlowe skreślenia curing wprowadzające w tekście pracy i dalsze na tej tematy stronach, materiały pracy. Powszechnie jest, aby tekst pracy oryginalnej by był podpisany na rodzajach: Wstęp, Materiał i metodyka, Wyniki, Omówienie wyników, Wstęp.
# STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. Komitake (Dept. of Hygiene, Faculty of Medicine, Fukuoka University, Fukuoka, Japan), N. Ishinok, and Y. Kodama</td>
<td>Faculty of Medicine, Kyushu University, Fukuoka, Japan</td>
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<table>
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<tr>
<th>Investigator's Phone No.</th>
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<tr>
<th>#: of Ref.'s</th>
<th>#: of Fig.'s</th>
<th>Language</th>
<th>(partial translation)</th>
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<td>24</td>
<td>28</td>
<td>Jpn</td>
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<table>
<thead>
<tr>
<th>Type &amp; duration of experiment</th>
<th>Purpose for study</th>
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<tbody>
<tr>
<td>Laboratory—indoors</td>
<td>to study effects of aircraft noise as a potential hazard to nearby residents</td>
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<tr>
<td>2 days tests per subject</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Description of test groups (subjects A, B, C, D, E)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9 female college students, average age 20.6, who did not live near an airport; (subjects were not sleeping at time of test)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Control of other agents</th>
<th>Statistical Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laboratory conditions—constant temp., and background noise (30 dBA)</td>
<td>(not translated)</td>
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</table>

<table>
<thead>
<tr>
<th>Noise Staging</th>
<th>CVS Response Measured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source: recorded aircraft noise</td>
<td></td>
</tr>
<tr>
<td>Spectral characteristics: high frequency—1000-2000 Hz</td>
<td></td>
</tr>
<tr>
<td>Noise level: average peak level=86.5 dBA, or 80 WCPNL (w/120 flyovers daily)</td>
<td></td>
</tr>
<tr>
<td>Length of exposure: continuously for 50 min. in AM, 100 min. in PM</td>
<td></td>
</tr>
<tr>
<td>#: of trials: 2 quiet &amp; 2 noise periods per subject (2 days total)</td>
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</table>

<table>
<thead>
<tr>
<th>Author's conclusions</th>
<th>Evaluation &amp; comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both jet aircraft noise and highspeed train noise should be controlled to less than WCPNL 80, since this level reduced people's aptitude on intellectual tests, causes mental fatigue, and may induce sustained tension of the sympathetic nervous system.</td>
<td>The abstract was written on the partially translated article (parts translated were those dealing with the experimental design and effects on pulse rate and fingertip pulse amplitude).</td>
</tr>
</tbody>
</table>

The effect of jet aircraft noise on mental fatigue, intellectual performance, pulse rate, and fingertip pulse amplitude was studied in 9 female college students, of average age 20.6 plus or minus 0.89 years. The students were of good health and did not live near an airport, where they might have had prior frequent exposure to jet airplane noise. The experiments were conducted in soundproof rooms, with a background noise of 30 dBA and a temperature of 26°C plus or minus 1.13°C. The subjects took a card-sorting test and Krapelin-Uchida's test under quiet conditions for 50 minutes in the morning and 100 minutes in the afternoon the first day. The second day, the same schedule was followed on the same subjects with the addition of continuous recorded jet aircraft noise equivalent to 120 flyovers (WEPNL 80) and an average peak level of 87 dBA. The noise was of high frequency having a peak of 1000 to 2000 Hz. Fingertip pulse amplitudes (of middle finger of left hand) and pulse rate were continuously monitored during the experiments. Degree of mental fatigue was measured before and after each session using the Cornell Medical Index of subjective psychophysiological complaints. Noise was associated with increased complaints related to hypertension. The intellectual tasks alone acted as stressors, causing increased pulse rate and decreased amplitude, which returned to normal levels on completion of the tasks. The fingertip pulse rate was greater before the subjects began the intellectual tests with the addition of jet noise. Noise also induced sustained tension of the sympathetic nervous system, since pulse amplitude decreased due to noise and did not return to normal levels after the intellectual tests were completed. The authors suggest that jet aircraft and highspeed train noise should be below WEPNL 80, due to the potential health hazards and effects on intellectual performance of this noise exposure level.
Partial Translation

STUDIES ON THE EFFECTS OF AIRCRAFT NOISE CAUSING MENTAL
FATIGUE DURING SOME INTELLECTUAL PERFORMANCE

Eizaburo Kunitsuke: Dept. of Hygiene, Faculty of Medicine,
Fukuoka University, Fukuoka

Naohara Ishinishi: Dept. of Hygiene, Faculty of Medicine,
Yasushi Kodama Kyushu University, Fukuoka

This is part 1 of a report of a study and investigation
carried out in 1963 and 1964 by the Medical Faculty of
Kyushu University relating to health hazards to residents
who were living in the vicinity of Itatsuke Air Base of
the U.S.A. in Fukuoka and were exposed to frequent aircraft
noises of jet planes.

Test Procedure

1. Subjects of the Test

Nine female students from the same division of a certain
public women's college were chosen as the subjects of the
tests. These female students did not live in the specific
air base vicinity and presented a healthy condition not
being affected by the exposure to the jet noise. Their
average ages were 20.5 ± 0.89 years old. A two-day conti-
nuous exposure test was scheduled for each subject avoiding
her menstrual period.

2. Exposure Test Room (insulated room) Conditions

Two insulated rooms were used for the test, i.e., one as a
noise exposure room and another as a fatigue examination room.
The insulated room consisted of: a 30cm thick outer wall,
a suspended sound-absorbing inner wall, double doors for
shutting the noise out and a non-echo type interior structure.
The back ground noise of the room was 30dB(A), and the room
temperature during the test periods was maintained at 26°C ± 1.13°C
by controlling the air. The subjects were under surveillance
from outside by monitoring TV. Communication between the
subjects and the investigators was made by interphone, while
fingertip pulse rates and pulse waves were measured by
polygraph from outside the room.
3. Conditions for Noise and Intellectual Tasks

The noise of flying jets (F-102, F-105) of the U.S. Air Force taped around Itatsuka Air Base was edited to produce an environment of continuous flying noise. The maximum value of the flying noise was between 95dBA and 85dBA and the average value was 87dBA, which was a high frequency range noise having its peak at 1000 and 2000 hertz. The time distribution of the noise exposure was 50 minutes in the morning and 100 minutes in the afternoon with a sufficient length of resting time in between. Each subject was committed to two days of intellectual tasks, i.e., the first day under noise-proof conditions and the second day under jet flying noise. The intellectual tasks consisted of Krapelin-Uchida's test using Uchida's method and a card sorting test.

Cornal Medical Index (C.M.I.) Questionnaire

Before and after the performance of the intellectual tasks, each subject was asked a total 81 questions relating to 9 items, C.M.I. and M - R out of the total questions regarding to 195 items of C.M.I., which was proposed by Fukamachi as the criteria to distinguish a neuropath.

According to the survey, the cases of complaints related to the cardiac blood vessel system, i.e., questionnaire item C, were 0.4% higher under a sound-proof environment than under a noisy environment before performing the tasks, and the cases of complaints were reduced by 0.4% under a sound-proof environment and by 0.3% under a noisy environment after completion of the tasks.

Fingertip Pulse Waves and Pulse Rates

A pulse meter was placed on the tip of the middle finger of the left hand before the start of the Krapelin test until the completion of the test in order to continuously measure pulse waves and pulse rates. The measured values were those obtained during first 15 seconds of every 10th minute, and the values were multiplied by 4 for the pulse rate while the amplitude of the pulse waves was indicated by the average value of the total wave height.

The impulse rate during Krapelin test under a sound-proof environment indicated an almost standardized fluctuation (Figure 4), where tension of the sympathetic nervous system during performing intellectual tasks and the relaxation of
the sympathetic nervous system during resting and after completion of the tasks were regularly observed. The maximum value was registered in 2-3 minutes after starting to perform the 1st series of tasks during the 50 minute test, while it was registered just before the completion of the tasks in the 2nd and 3rd series of tasks. Relating to the fingertip pulse rate under a noisy environment, the value before starting to perform the tasks was considerably large due to the noise, and the increase in pulse rate after starting the tasks, and the change in the pulse rate after completing the tasks were not as regular as seen under a sound-proof environment (Figure 5).

Figure (4) Key-1. 1st series of tasks  
2. 2nd series of tasks  
3. 3rd series of tasks  
4. before testing  
5. intermission  
6. after testing  

Change in Pulse Rate During Krapelin Test Under a Noisy Environment

![Graph](image)

Figure (5) Change in Pulse Rate During Krapelin Test Under A Sound-Proof Environment

Key-1. 1st series of tasks  
2. 2nd series of tasks  
3. 3rd series of tasks  
4. before testing  
5. intermission  
6. after testing

![Graph](image)
Referring to the fluctuations in amplitude of the pulse waves, they correspond to the pulse rate under the sound-proof environment (Figure 6), i.e., regular observation of tension and relaxation of the sympathetic nervous system as the tasks are being performed, minimum amplitude was registered during the first series of tasks and the maximum amplitude was registered during the last. Contrary to the above phenomena, under a noisy environment, a reduction in amplitude manifests strongly from before the beginning of the test (Figure 7), which remains all through the test allowing only slight increase of amplitude during resting and after completing the tasks.

Figure 6: Fingertip Pulse Wave Amplitude during Krapelin test under sound-proof environment

Key-1. 1st series of tasks
2. 2nd series of tasks
3. 3rd series of tasks
4. before testing
5. intermission
6. after testing

Figure 7: Fingertip Pulse Wave Amplitude during Krapelin Test Under Noisy Environment

Key-1. 1st series of tasks
2. 2nd series of tasks
3. 3rd series of tasks
4. before testing
5. intermission
6. after testing

Also, the fluctuation in amplitude is not regulated. An interesting point here is the fluctuation during the third series of tasks under the noisy environment. The values of the amplitude of the pulse waves are larger than in the preceding two performances, which suggests that the effect
of the noise lowered the tension of the sympathetic nervous system.

Comparing the amount of fluctuation and the maximum and minimum values of the pulse rate and pulse waves during the performance of tasks (Table 19), under the sound-proof environment, the maximum value and the amount of fluctuation increased mutually, whereas under the noisy environment, both the amount of fluctuation and the maximum value reached a minimum during performing the third series of tasks, indicating that the noise changed the tension/relaxation rhythm of the sympathetic nervous system which corresponded to the duration of the test and created a sustaining tensed state.

COMMENTS

The amount of noise that the subjects were exposed to at this time had an average peak level power of 86.5dB(A) and flying frequency of 120 planes, which is equivalent to 80 of WEPNL per day. When the subjects were continuously exposed to this level of noise while performing intellectual tasks, cortical and mental fatigue increased. Also tension of the sympathetic nervous system and impairment of aptitude for the tasks developed.

Conclusion

Among the subjective symptom of fatigue, "partial body pain" was most complained about under the noisy environment, all of which seemed to be related to the sympathetic nervous system.

It is appropriate for aircraft noise and super-express railway noise to be controlled under WEPNL 80, since this level of jet noise contributes to the increase in mental fatigue and the inhibition of aptitude for performing tasks.

Table 19: Fluctuation of pulse wave amplitude during Kreapelin Test

Key-1. 1st series of Kreapelin tasks
2. 2nd series of Kreapelin tasks
3. 3rd series of Kreapelin tasks
4. amplitude (cm)
5. under sound-proof environment
6. under noisy environment
7. maximum value
<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>3.</td>
<td>minimum value</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>difference</td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>maximum value</td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>minimum value</td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>difference</td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>maximum value</td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>minimum value</td>
<td></td>
</tr>
<tr>
<td>15.</td>
<td>difference</td>
<td></td>
</tr>
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</table>

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>0.14</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>1.51</td>
<td>1.57</td>
<td></td>
</tr>
<tr>
<td>0.68</td>
<td>0.66</td>
<td>0.18</td>
</tr>
<tr>
<td>0.83</td>
<td>0.79</td>
<td></td>
</tr>
<tr>
<td>1.60</td>
<td>2.61</td>
<td></td>
</tr>
<tr>
<td>0.49</td>
<td>0.37</td>
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<tr>
<td>1.13</td>
<td>1.14</td>
<td></td>
</tr>
<tr>
<td>2.18</td>
<td>1.49</td>
<td></td>
</tr>
<tr>
<td>0.66</td>
<td>0.73</td>
<td>0.70</td>
</tr>
<tr>
<td>1.22</td>
<td>0.70</td>
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</tbody>
</table>
Studies of the Effect of Aircraft Noise causing Mental Fatigue during some Intellectual Performance

Eitaro Kuniike
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Noburu Ishihara, Yasushi Kedem
Department of Hygiene, Faculty of Medicine, Kyushu University, Fukuoka

Studies of mental fatigue caused by exposure to the aircraft noise was carried out experimentally on 8 male students engaged in some intellectual performance. The impact-echoed noise of the flying jet engine was controlled from 85(dB(A)) to 90(dB(A)) in the peak level. The subjects were exposed continuously to the noise during 60 minutes and 90 minutes, respectively, in an isolated room provided from general noise noise.

Through the noise exposure period, the subjects performed intellectual tasks, that is, the reaction time and the reaction time. At the same time, pulse rate and pulse waves of their fingers were measured. Before and after each exposure, the mental fatigue was measured by the critical flicker frequency (C.F.F.), the pulse rate, and the blood pressure of the multiple performance test (M.P.T.). And the knowledge of subjective sensations of mental fatigue were collected by the questionnaire, General Medical Index (G.M.I.) were used for psychophysiological complaints.

The control data of each subject were obtained from the same experiment except noise.

The following conclusions were derived from observations. The grade of mental fatigue increased in the case of the noise exposure noise than in the case of the noiseless situation. In C.F.F., fatigue increased 25% under noise exposure of 60 minutes, 37% under that of 90 minutes. In C.N. and M.P. as well as C.P.F., the same gradient between the noise exposure and fatigue were found. Furthermore, fatigue showed itself more clearly in G.N. than in C.D.F., namely, the time of performance of the time required for each identifying exceeded the critical level of mental fatigue, while the rate of mental fatigue in C.F.F. was within the normal or allowable range.

The results of the questionnaires of subjective sensations of fatigue and of the G.M.I. in the case of the noise exposure showed no remarkable differences compared with those of the noiseless situation. But, it was found that more complaints of sensation of fatigue increased slightly during noise exposure compared with those in the noiseless situation. These complaints were the symptoms probably caused by the sympathetic nervous system. Moreover, from the observation of the pulse rate and blood pressure, it was considered that a biological side of the relaxation of the sympathetic nervous system simultaneously when they were exposed to the mental task.

From the observation mentioned above, it is considered that there is a possibility of the influence of the learning effectiveness in school children when they are exposed to the aircraft noise such as
I. 論 文

総合的の実験に当たっては多くの研究がある。そのなかで、個々の心身共に、社会的に、また生活についての多くの研究が行われている。

これについて、改善点として特殊なつまりであるところは、

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これに関して、改善点として特殊なつまりであるところは、

これに関

II. 実験方法

1. 実験方法

実験方法における一例は以下の通りで、いずれも高音を含む音圧レベルでの実験を行っており、実験結果を示すための図は以下にて示す。

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実験方法における一例は以下の通りで、いずれも高音を含む音圧レベルでの実験を行っており、実験結果を示すための図は以下にて示す。

3. 実験方法（実験方法）

実験方法における一例は以下の通りで、いずれも高音を含む音圧レベルでの実験を行っており、実験結果を示すための図は以下にて示す。
図1  結果のTime Table

1. レターテータ分析

分析は以下の通りである。（データ1）は分析の選定
した20名（10名の平均）と、普通の10名（平均）の
平均を比較したものです。（T検定）で有意水準
を1.0%と設定した結果）

<table>
<thead>
<tr>
<th>作業時間</th>
<th>平均作業量</th>
<th>標準差</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5日間</td>
<td>2.1±1.85</td>
<td>2.1</td>
</tr>
<tr>
<td>0.5日間</td>
<td>2.2±1.90</td>
<td>2.2</td>
</tr>
</tbody>
</table>

注：各項目にそれぞれの作業者の結果については、統計的な
解析が行われ、有意差を確認した結果を示しています。
表2 訪問時、検査前の健康状態、作業環境の発熱基準

<table>
<thead>
<tr>
<th>H</th>
<th>S</th>
<th>Y</th>
<th>N</th>
<th>Y</th>
<th>T</th>
<th>M</th>
<th>K</th>
<th>V</th>
<th>H</th>
<th>N</th>
<th>K</th>
<th>N</th>
<th>T</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>第1作業</td>
<td>1.19</td>
<td>1.15</td>
<td>1.17</td>
<td>1.05</td>
<td>1.22</td>
<td>1.14</td>
<td>1.17</td>
<td>1.21</td>
<td>1.16</td>
<td>1.17</td>
<td>0.009</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>第2作業</td>
<td>1.21</td>
<td>1.06</td>
<td>1.11</td>
<td>1.10</td>
<td>1.08</td>
<td>1.07</td>
<td>1.04</td>
<td>1.06</td>
<td>1.05</td>
<td>1.04</td>
<td>0.062</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>第3作業</td>
<td>1.12</td>
<td>1.04</td>
<td>1.01</td>
<td>1.02</td>
<td>1.00</td>
<td>1.04</td>
<td>1.01</td>
<td>1.02</td>
<td>1.01</td>
<td>1.01</td>
<td>0.041</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>第4作業</td>
<td>1.14</td>
<td>1.05</td>
<td>1.15</td>
<td>1.20</td>
<td>1.06</td>
<td>1.09</td>
<td>1.03</td>
<td>1.01</td>
<td>1.01</td>
<td>1.07</td>
<td>1.09</td>
<td>0.071</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>第5作業</td>
<td>1.15</td>
<td>1.06</td>
<td>1.11</td>
<td>1.00</td>
<td>1.09</td>
<td>1.00</td>
<td>1.04</td>
<td>1.04</td>
<td>1.01</td>
<td>1.05</td>
<td>1.07</td>
<td>0.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>第6作業</td>
<td>1.08</td>
<td>0.83</td>
<td>0.83</td>
<td>0.86</td>
<td>0.90</td>
<td>0.94</td>
<td>0.97</td>
<td>0.95</td>
<td>0.98</td>
<td>0.99</td>
<td>0.067</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* 平均値±標準偏差

表3-1 訪問時、検査前の作業環境の発熱基準

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.17</td>
<td>1.09</td>
<td>1.03</td>
<td>1.07</td>
<td>1.05</td>
<td>1.01</td>
<td>1.07</td>
<td>1.09</td>
<td>0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* 発熱基準判定基準

表3-2 応診結果の1.16以下の基準

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
<td>0.9</td>
</tr>
</tbody>
</table>

* 発熱基準判定基準

表3-3 作業環境による発熱基準の選下

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.17</td>
<td>1.09</td>
<td>1.03</td>
<td>1.07</td>
<td>1.05</td>
<td>1.01</td>
<td>1.07</td>
<td>1.09</td>
<td>0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* 発熱基準判定基準

(注) **1.021、1.013の基準 **
<table>
<thead>
<tr>
<th>番号</th>
<th>種類</th>
<th>項目</th>
<th>数値1</th>
<th>数値2</th>
<th>数値3</th>
<th>数値4</th>
<th>数値5</th>
<th>数値6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>K</td>
<td>17</td>
<td>0.46</td>
<td>0.35</td>
<td>0.56</td>
<td>0.35</td>
<td>0.46</td>
<td>0.35</td>
</tr>
<tr>
<td>2</td>
<td>K</td>
<td>22</td>
<td>0.51</td>
<td>0.42</td>
<td>0.45</td>
<td>0.42</td>
<td>0.51</td>
<td>0.42</td>
</tr>
<tr>
<td>3</td>
<td>K</td>
<td>30</td>
<td>0.52</td>
<td>0.43</td>
<td>0.48</td>
<td>0.43</td>
<td>0.52</td>
<td>0.43</td>
</tr>
</tbody>
</table>

**注記:**

1. 本表は実験データに基づいて作成されたもので、正確性を保証するものではない。
2. 数値の関係性についての詳細は、後日発表予定。
3. 未だ検討中であるが、今後、さらなるデータを収集し、詳細を公表予定。
図4 作業成績の時間の変動

作業の作業成績が、時間の変動に合わせて大きな変化が見られる。時間の変動が、作業の作業成績に大きな影響を及ぼすことが示唆されている。

図5 作業成績の時間の変動（100分とした場合）

<table>
<thead>
<tr>
<th>タイム</th>
<th>50分作業</th>
<th>100分作業</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>60</td>
</tr>
</tbody>
</table>

*（）は作業の作業成績が時間によって変動することが示唆されている。

100分作業における作業成績の変動が時間によって大きく変動する傾向があることが示唆されている。
<table>
<thead>
<tr>
<th>表14</th>
<th>作業区分の N.P. 関値分布の差異</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>作業区分</td>
</tr>
<tr>
<td>50分作業</td>
<td>8.25 ± 0.058</td>
</tr>
<tr>
<td>50分作業</td>
<td>8.18 ± 0.051</td>
</tr>
<tr>
<td>100分作業</td>
<td>0.19 ± 0.053</td>
</tr>
<tr>
<td>100分作業</td>
<td>0.18 ± 0.057</td>
</tr>
</tbody>
</table>

* なお、作業区分は以下の通り。

<table>
<thead>
<tr>
<th>表15</th>
<th>作業区分の分布の差異の検定の結果</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>作業区分</td>
</tr>
<tr>
<td>A</td>
<td>自然</td>
</tr>
<tr>
<td>B</td>
<td>自然</td>
</tr>
<tr>
<td>C</td>
<td>無自然</td>
</tr>
</tbody>
</table>

* なお、上記の結果は統計学的に有意である。
図4 元々、クレパンによるデータのグラフ

図5 各々の実験で得られたクレパンの結果を示す。

表1 クレパンによるデータのまとめ

<table>
<thead>
<tr>
<th>実験条件</th>
<th>最大値</th>
<th>平均値</th>
</tr>
</thead>
<tbody>
<tr>
<td>実験1</td>
<td>1.50</td>
<td>1.57</td>
</tr>
<tr>
<td>実験2</td>
<td>0.95</td>
<td>0.98</td>
</tr>
</tbody>
</table>

IV. 論巻

学習方法については、クレパンによる実験で、以下の結果が得られているが、学

実験1においては、クレパンの使用により、結果は良好であったが、実

験2においては、クレパンの使用により、結果は悪化した。この結果は、クレ

パンの効果の定義が問題であることを示している。これにより、クレ

パンの使用が必要である。
この文章は日本語で記載されているため、翻訳は対応しておりません。
言語は自然言語で、文章の意味を把握するための理解ガイドラインが提供されている。文章の内容を適切に説明すると、以下のようになる。

また、文章の内容は日本語であり、特に英語の文脈を必要としない場合、自然に読むことができる。
ルールは守られています。目次は、以下のとおりです。
1. はじめに（1960-1971）
2. はじめに（1972-1973）
3. はじめに（1974-1975）
4. はじめに（1976-1977）
5. はじめに（1978-1979）
7. はじめに（1982-1983）
8. はじめに（1984-1985）
9. はじめに（1986-1987）
10. はじめに（1988-1989）
11. はじめに（1990-1991）

文献
1. 西川浩司：論文（1960-1971）
2. 西川浩司：論文（1972-1973）
3. 西川浩司：論文（1974-1975）
4. 西川浩司：論文（1976-1977）
5. 西川浩司：論文（1978-1979）
7. 西川浩司：論文（1982-1983）
8. 西川浩司：論文（1984-1985）
9. 西川浩司：論文（1986-1987）
10. 西川浩司：論文（1988-1989）
11. 西川浩司：論文（1990-1991）

(終了 1989年5月)
## STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

### Principal Investigator(s):
- A.Z. Martysako
- V.V. Lipovoy

### Institution and address where research was performed:
Institute of Labor Hygiene and Professional Illnesses; USSR

### Investigator's Phone No. Sponsoring Organization:
same as above

### Citation:
Martysako, A.Z. and V.V. Lipovoy, An estimate of the total time of individual noise effects in hygienic evaluation of intermittent noises. Gig. Truda i Prof. Zabol (8):15-18, 1972

### Type & duration of experiment:
- laboratory--acoustically isolated chamber

### Purpose of study: to compare the effects of continuous v. intermittent noise on fingerpulse amplitude, pulse rate, brain function, and auditory threshold

### Description of test groups (subjects, N, age, sex, etc.)
- 20 healthy 16-25 year old men

### Control of other stressors:
laboratory conditions

### Statistical Methods used but not specified

### Noise Stimulus
- source: not specified
- spectral characteristics: low frequency--250 cycles/sec. (average)
- noise level: 110 dB (low frequency)
- 105 dB (high frequency)
- length of exposure: 1 hour (total time equal for continuous and intermittent noise)
- 6 of trials: 4 series; 20 observations each (1 hr. noise, 1 or 3 hrs. quiet per series)

### CVS Response Measured:
- pulse rate
- fingerpulse amplitude (index finger)

### Nonauditory effects:
- fingerpulse amplitude--greater decrease with CVS; intermittent noise than with continuous noise. Pulse rate--decreased pulse rate w/both continuous & intermittent noise

### Other:
- reaction time and accuracy on a simple task decreased due to noise to a greater degree with intermittent noise.

### Author's conclusion:
Continuous noise decreased hearing sensitivity to a greater degree than intermittent noise. Greater effects on the vascular and nervous systems were produced by intermittent noise. The authors feel that the main variable is the combination of noise and quiet used, not the total noise exposure time.

### Evaluation & comments:
The number of subjects in each of the 4 parts of the experiment was not defined.

The effects of intermittent and continuous noise on hearing sensitivity, the nervous system, and the cardiovascular system were studied in 20 healthy men, 18 to 25 years old. The nervous system effects were tested using the subjects' reaction time to a simple task. Cardiovascular functioning was measured using the pulse rate and the fingerpulse amplitude. The experiment included 4 parts, all of which were done in a sound-isolated room. The first 2 parts studied low frequency (medium 250 cycles per second) noise at a level of 110 dB for a total of 1 hour noise exposure and 2 hours quiet. Part 1 involved a 1 hour continuous noise dose, and part 2 consisted of 2½ min. noise and 5 min. quiet alternately for 3 hours. Parts three and four of the experiment used a noise level of 105 dB at a high frequency (medium frequency 200 cycles per second) for a total time of 1 hour noise and 1 hour quiet. A continuous noise dose was used in part three; ½ minute noise was alternated with ½ minute of quiet in part four. Continuous noise had a greater effect on hearing sensitivity, whereas intermittent noise had a stronger effect on the nervous system and cardiovascular functioning. Subjects made more errors on the simple task during the intermittent noise of high frequency than with high frequency continuous noise; no difference in number of errors was found with low frequency noise. Intermittent noise, especially of high frequency, affected the fingerpulse amplitude response to noise. The decrease in amplitude was greater with 50 minutes of intermittent noise (52% of the original amplitude) than with the same exposure to continuous noise (72% of the original amplitude). The pulse rate decreased by 2 or 3 beats with both continuous and intermittent noise. The total noise exposure time may not be as important as the combinations of noise and quiet in producing certain nonauditory physiological effects.
AN ESTIMATE OF THE TOTAL TIME OF INDIVIDUAL NOISE EFFECTS
IN HYGIENIC EVALUATION OF INTERMITTENT NOISES

By A.Z. Marinyako and V.V. Lipovoy

Institute of Labor Hygiene and Professional Illnesses

(Arrived at the editor July 20, 1971).

An analysis of the literature concerning the study of the influence of intermittent acting noises on the organism shows that in one case the investigators observed a more expressed influence in the case of an intermittent effect, and in the other case, the intermittent effect of noise caused the same effect or to a lesser degree a biological effect compared with continuous noise. Some authors (Poth and Weinberg) believe that the degree of harmfulness of intermittent noises together with the calculation of the level and of the spectral makeup is determined by producing the general time of their effect on acoustical energy, that is to say, in the opinion of the indicated investigators, the [illegible] amount of effective acoustical energy is harmful to the same extent depending on how this energy is distributed in time. Meanwhile, according to the data of Schröder and Rempt, stable noise causes greater shifts on the part of some physiological functions, in particular hearing, than intermittent noise (other conditions being equal), if the time of effect of the stable noise and the total time of the intermittent noise are the same.

In connection with what has been stated, we are confronted with the problem of studying the characteristics of the influence of some forms of intermittent noise compared with stable noise with the same total effect time of them. The studies were carried out in a sound-insulated chamber with 20 practically healthy people of the male sex aged 18-25. There was a series of 4 with 20 observations in each. In all the series, the people being studied were subjected to the effect of noise for the course of 1 hour.

In the first series, we studied the influence of low frequency (maximum acoustical energy in the octave band with a medium geometric frequency of 250 cycles per second) stable noise with a level of 110 decibels. In the second series, we established the influence of noise with the same level and spectrum, but acting intermittently: the noise period of effect of 2 1/2 min alternated with 5 minute intervals. Such a character of the noise exists in some operational regimes of a vibrational area according to the condensation of the concrete. The general time of the experiment in this series of investigations amounted to 3 hours (total time of the noise 1 h and the time of the pause 2 h). In the IIIrd series we studied
the influence of high frequency (maximum acoustical energy in the octave band with medium geometric frequency 200 cycles per second) stable noise with a level of 105 decibels. In the IVth series, we evaluated the influence of intermittent noise, where the half minute high frequency sounds with a level of 105 decibels alternated with half minute intervals. Noise with such parameters is noted with the hydraulic study of pipes. The time of the experiment in the given series of investigations is 2 h (period of the noise 1 h and the time of the pause 1 h).

With the subjects, we studied the functional state of the auditory equipment, the cortex of the brain and some functions of the heart-vessel system (tone of the vessels, frequency of the pulse). The auditory sensitivity was determined by an audiometer at tones of 500, 1000, 2000, 4000 and 6000 cycles per second by studying the sound conductivity of the air.

The study of the functional state of the brain cortex was carried out with the aid of an apparatus for studying advanced nerve activity, the principle of effect of which is based on presenting stimulation to the subject with a different wave characteristic and obtaining the corresponding response to it.

On the front panel of the instrument there are two rows of buttons (8 buttons in each) of different colors and a screen on which colored signals appear in a determined sequence at a rate of 22 to 110 per minute which light up and go out automatically. With the appearance on the screen of the same or different colored signal-stimulation, the subject must press the button of the corresponding color. With a correct and well-timed reaction (up to the appearance of the following stimulation), a signal confirmation is given along with the signal stimulation. The sequence of the presentation of stimulations is determined experimentally with the aid of 4 programs (according to 8 colored signals in each). The maximum rhythm of the delivered signals is established for each subject at which his responses contain the minimum number of errors. For the majority of those studied, 60 presentations per minute is the maximum rhythm. The tone of the vessels is determined with the aid of a twin-channel, plethysmograph. The plethysmogram is recorded with the index finger. When analyzed, the plethysmograph is considered to be the amplitude of the volumetric changes and the amplitude of the pulse strokes.
The results of audiometric studies showed that despite the similar total amount of acoustical energy, the stable noises caused a more expressed decline of hearing sensitivity, than intermittent noise. So, after the effect of low-high frequency noises with levels of 110 and 105 decibels, a decline of hearing in the frequency of 4000 cycles per second was noted, corresponding to 24 and 26 decibels. After the effect of the intermittent noises, a drop of 15 and 14 decibels was noted. It should be noted that after the effect of the stable noises, we noted a decline of hearing not only in the frequency of 4000 cycles per second, but at adjacent frequencies—3000 and 6000 cycles per second (on the average of 20 and 17 decibels). After the effect of intermittent noises, the decline of auditory sensitivity at the indicated adjacent frequencies was less expressed (10-13 decibels). The fact that the time reduction of hearing was noted at tones adjacent to the tone 4000 cycles per second is evidence of the great fatigue of the auditory analyzing equipment with the effect of stable noise compared with intermittent noise.

The results of the study of advanced nerve activity show that in the case of the effect of stable as well as of intermittent noises there was a decline in the rate of the nerve processes in the case of those studied, certainly increasing the amount of erroneous responses to the colored stimulations. However, a more expressed tendency to a worsening of the indicated characteristics was observed after the effect of intermittent noises. So, after the effect of high-frequency, intermittent noises with a level of 105 decibels, the subjects made 2.7±0.26 errors where the original amount was 1.9±0.18 errors. Stable noise caused an increase of the erroneous responses up to 2.5±0.27 errors compared with an original value of 2.0±0.17 errors.

After the effect of the low frequency, stable and intermittent noises with a level of 110 decibels, the amount of erroneous responses increased to the same degree, in the case of stable noise from 3.8±0.21 to 5.4±0.43 and with intermittent noise from 3.0±0.27 to 4.6±0.29.

A rather notable difference in the effect of stable and intermittent noises (especially with high frequency components) was observed in the study of the tone of vessels (see figure). It is seen on the drawing that with a 10 minute effect of stable and intermittent noises, the amplitude of the plethysmogram amounted to 65 and 62% correspondingly of the original level. In a 50 min exposure to stable noise, which constitutes the same time of the general total effect of intermittent noise, the difference in the amplitude changes of the plethysmogram was more noticeable: in the case of a stable noise, its value amounted to 72%, in the case of an intermittent noise, it amounted to 52%. The observed difference in amplitudes is statistically reliable.
Background of the 1st min. 50 min/1 h

Change in the amplitude of the finger plethysmograph

According to the axis ordinate—amplitude of the plethysmograph (in %); according to the axis of the abscissa, effect time of the noises; 1—stable noise; 2—intermittent noise.

After a one hour effect of the stable and intermittent noises, we observed a tendency toward reducing the frequency of the pulses on the average of 2-3 per minute. It is not thought possible to explain the dependence between the character of the effective noises and the degree of reduction of the pulse.

Thus, the investigations which were carried out provide a basis for thinking that the intermittent effect of noises causes a different reaction compared with stable, despite the similar amount of acoustical energy, perceived by the hearing organ. So, for the auditory analyzer, the distribution of acoustical energy in time with an intermittent noise effect turns out to be positive. Evidently, the comparatively long pauses between noise facilitate auditory sensitivity and an increase in the adaptation processes. The fact that after the effect of intermittent noises, the functional state of the brain cortex is changed to a large degree is probably explained by the fact that intermittently the active noises are subjectively perceived as more unpleasant, and due to this may exert a greater effect on the mental activities of the man, which in turn reflects the condition of the higher nerve activity.

The more expressed vessel reaction to the effect of intermittent noise evidently may explain the difficulty of working out an adaptation of the vessel system to individual intermittent sounds.
Taking into account the results of the present studies, and also the studies carried out by us and other fellow workers in our laboratory in recent times (A.A. Men'shov et al.), it can be expected that with the hygienic characteristic of the intermittent effective noises in each concrete case, one must mainly take into account the time combinations of the "noise-pause" complex, and not the total time of effect of the noise.

Literature

AN ESTIMATE OF THE TOTAL TIME OF INDIVIDUAL NOISE EFFECTS IN HYGIENIC EVALUATION OF INTERMITTENT NOISES

A. Z. Martingale, W. V. Lyvoy

Summary

The effect on the human organism of static noise (low-frequency of 110 db and high-frequency of 105 db) and of the same, but intermittent noise was studied in a soundproof chamber. Low-frequency soundings of 5 min, 30 sec each alternated with 3 minute long intervals. Half-minute long high-frequency soundings alternated with half-minute long intervals. In all cases the sum total of the exposure to the effect of sound lasted one hour. In persons undergoing examination the authors determined time shifts of the auditory thresholds, functional state of the brain cortex, vascular tone and pulse rate. The effect of static noise was found to produce more pronounced changes in the auditory function, whereas the local of the higher nervous and vascular systems the shifts proved greater under the effect of intermittent noise.

The work of the emergency medical aid physicians entails considerable mental and emotional stress, a high sense of responsibility and is distinguished by the complexity of the functions to be performed. The authors have set the task to study the working conditions of the emergency aid physicians, to elucidate charges of their performance capacity throughout the 24 hour long stay on duty. The functions of the central, vegetative and cardiovascular systems were investigated to measure the degree of fatigue. An analysis of the resultant information enabled it to establish the presence of fatigue and emotional stress in physicians of the emergency medical aid consequent to performance of their duties and also to clear up certain regularities in the dynamics of these processes taking place in such system under study. A subjective sensation of fatigue made itself felt simultaneously with initial signs of fatigue, but, while the fatigue in some systems of the organism somewhat decreased by the end of the work-day under the effect of some rest, the sensation of general fatigue steadily increased. In emergency medical aid physicians with a long service record no manifestations of overstrain were observed.

УЧЕТ СУММАРНОГО ВРЕМЕНИ ОТДЕЛЬНЫХ ШУМОВЫХ ВОЗДЕЙСТВИЙ ПРИ ГИГИЕНЕЧЕСКОЙ ОЦЕНКЕ ПРЕРЫВАЕМЫХ ШУМОВ

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Анализ литературы, касающейся изучения влияния на организм прерывного действующих шумов, показывает, что в одном случае исследователи находили более выраженные изменения при прерывистом воздействии, а другой — прерывистое воздействие шума оказывало одинаковый или в меньшей степени биологический эффект по сравнению с непрерывным. Некоторые авторы (Poth и Weinberg) считают, что степень артериального давления в прерывистых шумах параллельна с учетом уровня и спектрального состава определяется произведениями общего времени их действия на акустическую энергию, так что уменьшение упомянутых исследователей, равно как и падение воздействующей акустической энергии различными образцами шумов значительно от того, как эта энергия распределяется по времени. Между тем, по данным Schröder и Rempel, стабильный шум вызывает большие сдвиги со стороны некоторых физиологических функций, в частности слуховой, чем прерывистий (при крайних разных условиях), и при воздействии стабильного шума и суммарное время прерывистого одинаково.
В связи с изложенными перед нами была поставлена задача изучить особенности влияния некоторых видов прерывистых шумов по сравнению со стабильными при одинаковой суммарной длительности действия их. Исследования проводились в звукоизолированной камере с 20 практиков здоровых лиц мужского пола в возрасте 18—25 лет, всего 4 серии исследований по 20 наблюдений в каждой. Во всех сериях исследуемые лица подвергались воздействию шума в течение 1 час.

В 1 серии изучали влияние низкочастотного (максимум звуковой энергии в октавной полосе среднегеометрической частотой 250 гц) стабильного шума уровнем 110 дБ. Во II серии влияние шума с таким же уровнем и спектром, но действовавшего прерывисто: шумовые воздействия продолжительностью 2/3 час. чередовались с 5-минутными перерывами. Такой характер шума бывает при некоторых режимах работы вибрационных площадок при уплотнении бетона. Общее время опыта в этой серии исследований составляло 3 часа (суммарное время шума 1 час и время пауз 2 часа). В III серии исследовали влияние высокочастотного (максимум звуковой энергии в октавной полосе со среднегеометрической частотой 2000 гц) стабильного шума уровнем 105 дБ. В IV серии оценивали влияние прерывистого шума, при котором понижение высокочастотных звуков упражнением 105 дБ чередовалось полуминутными паузами. Шум с такими параметрами отмечается при гидравлическом испытании труб. Время экспериментов в данной серии исследований 2 часа (длительность шума 1 час и время пауз 1 час).

У исследуемых лиц изучали функциональное состояние слухового анализатора, коры головного мозга и некоторые функции сердечно-сосудистой системы (токсус сосудов, частота пульса). Слуховую чувствительность определяли аудиометром на тонах 500, 1000, 2000, 4000 и 6000 гц путем исследования воздушной звукопроводимости.

Изучение функционального состояния коры головного мозга проводили с помощью прибора для исследования высшей двигательной и сенсорной областей, принцип действия которого основан на превращении исследуемого раздражителя в различных волокнах характеристикой и получении соответствующего ответа на них.

На передней панели прибора находятся два ряда кнопок (по 6 кнопок в каждом) различного цвета и экран, на котором в определенной последовательности и со скоростью от 22 до 110 в минуту появляются световые сигналы, которые загораются и гаснут автоматически. При появлении на экране того или иного светового сигнала-раздражителя исследуемый должен нажать кнопку соответствующего цвета. При правильной и своевременной реакции (до появления следующего раздражителя) рядом с сигналом-раздражителем зазвучит сигнал-подтверждение. Последовательное раздражение раздражителем определяется экспериментатором при помощи 4 программ (по 8 цветовых сигналов в каждой). Для каждого исследуемого устанавливали максимальный ритм подаваемых раздражителей, при котором его ответы содержали минимальное количество ошибок. Для большинства исследуемых 60 предъявлений в минуту являлись максимальным ритмом. Тонус сосудов определяли с помощью плетизмографического чередующегося пульсового рефлекса. Плетизмограмму записывали с указательного пальца. При анализе плетизмограммы учитывали амплитуду объемных изменений и частоту пульсового удара.

Результаты аудиометрических исследований показали, что несмотря на интенсивность звуковой энергии, стабильные шумы вызывали более выражаемое снижение слуховой чувствительности, чем прерывистые. Так, после воздействия низкочастотных шумов с уровнями 110 и 105 дБ снижение слуха на частоте 4000 гц отмечалось соответственно на 24 и 26 дБ; после воздействия прерывистых — на 15 и 14 дБ. Следует отметить, что после воздействия стабильных шумов отзе-
наблюдалось после воздействия прерывистых шумов. Так, после воздействия высокочастотного прерывистого шума уровень 105 дБ искажений составлял 2,7±0,26 ошибок при исходном количестве 1,9±0,18 ошибок. Стабильный шум вызывал увеличение ошибочных ответов до 2,3±0,27 ошибок по сравнению с исходным значением 2,0±0,17 ошибок.

После воздействия низкочастотного стабильного и прерывистого шумов уровень 110 дБ количество ошибочных ответов возрастило в одинаковой степени при стабильном — с 3,8±0,21 до 5,4±0,43 и при прерывистом — с 3,0±0,27 до 4,9±0,29.

Довольно заметная разница в действиях стабильных и прерывистых шумов (особенно в низкочастотных составляющих) наблюдалась при исследовании тонуса сосудов (см. рисунок). На рисунке видно, что на 1-й минуте действия стабильного и прерывистого шумов амплитуда пульсограмм составляла соответственно 63 и 62% исходного уровня. На 50-й минуте экспозиции стабильного шума, что составляет такое же время общего синхронного действия прерывистого шума, различия в изменении амплитуды пульсограмм была более значительна: при стабильном шуме ее величина составляла 72%, при прерывистом — 52%. Наблюдаемое различие в амплитудах статистически достоверно. После одновременного воздействия стабильных и прерывистых шумов наблюдалась тенденция к уменьшению частоты пульса в среднем на 2—3 в минуту. Выяснить зависимость между характером воздействующих шумов и степенью урежения пульса не представлялось возможным.

Таким образом, проведенные исследования дают основание считать, что прерывистое действие шума вызывает несколько иные реакции по сравнению со стабильным, несмотря на одинаковое количество звуковой энергии, воспринимаемой органом слуха. Так, для слухового анализатора распределение звуковой энергии во времени при прерывистом шумовоздействии сказывается положительно. По-видимому, относительно длительные межшумовые паузы способствуют восстановлению слуховой чувствительности и улучшению процессов адаптации. Этот факт, что после воздействия прерывистых шумов функциональное состояние коры головного мозга изменяется в большей степени, вероятно, объясняется тем, что прерывисто действующие шумы субъективно воспринимаются как более неприятные, а отсюда могут оказывать более воздействие на психическую деятельнос.
ность человека, которая в свою очередь отражает состояние высшей нервной деятельности.

Более выраженную сосудистую реакцию на воздействие прерывистого шума, по-видимому, можно объяснить трудностью выработки адаптации сосудистой системы к отдельным прерывистым звукам.

Принимая во внимание результаты настоящих исследований, а также исследования, проведенные нами и другими сотрудниками нашей лаборатории в последнее время (А. А. Меншиков и соавт.), можно полагать, что при гипокинетической характеристике прерывистых действительных шумов в каждом конкретном случае следует учитывать таким образом временные компоненты комплекса «шум — пауза», а не сумму общее время воздействия шума.

Литература

Schroeder K., Rempel E., Lärmbelästigung, 1962, Bd 5—6, S. 142.
SECTION 16
<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tr>
<td>T. Matoba (Dept. of Medicine, Kurume University School of Medicine, Kurume 830), H. Kusumoto, H. Osuda, T. Kocori, H. Kuwabara, H. Takanatsu</td>
<td>Laboratory of Clinical Physiology, Yufuin Kosei Nenkin Hospital</td>
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**Citation:** Matoba, T., et al. Digital plethysmographic responses to auditory stimuli with vibration disease. Tohoku J. Exp. Med. 115(4):385-392, 1975

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<tr>
<th>Type &amp; duration of experiment</th>
<th>Purpose for study</th>
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<td>type: laboratory-in sound-proof room</td>
<td>to study effects of noise on sympathetic nervous system in workers with vibration disease</td>
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**Description of test groups (subjects, n, etc.):**
- control group: 15 healthy men aged 43.8 ± 3.1 years; average blood pressure 123.9 ± 12.5 over 74.9 ± 8.8 mm. mercury.
- test group: 82 male patients with vibration disease who worked as chain-saw operators; average age 48.6 ± 7.3 yrs.; average blood pressure 129.3/77.3

**Control of other stressors:** Chain-saw workers were exposed to cold temp., noise, and vibration apparatus in their work.

**Noise Stimulus:**
- source: chain-saw noise recordings introduced through headphones
- spectral characteristics: not given
- noise level: 98-102 dB
- length of exposure: 10 sec.
- # of trials: not specified

**Statistical Methods:**
- Test for significance used

**CVS Response Measured:**
- digital plethysmogram (finger pulse amplitude)

**Nonauditory effects:**
- healthy subjects: immediate decrease in CVS; digital plethysmographic amplitude due to noise; recovery to normal amplitude in 30 sec. men w/vibration disease: slower response and recovery times
- Other: not studied

**Author's conclusions:** Vibration disease is associated with impaired autonomic nervous system response. Digital plethysmographic changes due to auditory stimuli can serve as indicators of autonomic activity; as a diagnostic tool.

**Evaluation & comments:** The finger pulse amplitude data for the healthy controls illustrates the effects of noise on blood vessels (vasoconstriction) very clearly—graphs are included.

Finger pulse amplitude responses were compared in patients with vibration disease and in healthy subjects. The healthy subjects were 15 males from the Japanese Self-Defense Force aged 43.8 plus or minus 3.1 years and with average blood pressures of 123.9 plus or minus 12.5 over 74.9 plus or minus 6.8 mm mercury. The group with vibration disease consisted of 82 male chain-saw operators aged 48.8 plus or minus 7.3 years and with average blood pressures of 129.3 plus or minus 12.5 over 77.5 plus or minus 10.5 mm mercury. Healthy subjects were selected using the Cornell Medical Index. Digital plethysmograms were obtained in response to recorded chain-saw noise introduced through headphones in a soundproof room. The noise dose was from 98 to 102 dB for 10 seconds. Post-stimulus recovery was monitored by digital plethysmogram for up to 60 seconds from the beginning of the noise. The noise produced an immediate decrease in amplitude of the plethysmograms in the healthy subjects. The amplitude usually returned to normal within 30 seconds in these subjects. The patients with vibration disease had both poor responses to the noise and slower recovery times—they were still abnormal at 60 seconds. Individual differences in the plethysmograms were considerable among all the subjects. The response of the small arteries in the fingers to noise is under control of the autonomic nervous system. When a drug (tolazoline hydrochloride) that blocks autonomic nervous system receptors was given, the finger pulse responses to noise were poor. Digital plethysmograms in response to noise can be used as indicators of the functioning of the autonomic nervous system.
Digital Plethysmographic Responses to Auditory Stimuli in Patients with Vibration Disease

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Digital plethysmographic responses to auditory stimuli in 15 healthy men and 82 patients with vibration disease were analyzed in order to clarify the functional conditions of autonomic nervous system in this disease. The auditory stimuli given to healthy men caused a rapid decrease in the amplitude of the plethysmograms. After cessation of the auditory stimuli the decreased amplitude recovered to the control value within 30 sec. In the patients with vibration disease, however, the recovery of the decreased amplitude was delayed. The plethysmographic changes in the patients with vibration disease were divided into 4 types: normal (N), intermediate (I), delayed (D) and poor response (P) types. Each type of I, D and P was altered to type N by treatments consisting of therapeutic exercises, but spring ales and so on. All healthy men showed type N. There were no significant differences between the time courses of the recovery of the plethysmographic changes and the amplitudes of the plethysmograms before the auditory stimuli. The results obtained seem to indicate that the autonomic nervous system in the patient with vibration disease is in disorder, and that the digital plethysmography with auditory stimuli is instrumental to detect the functional changes in the autonomic nervous system.

A digital plethysmography has been reported to be able to detect the condition of the sympathetic activity in man (Aclmer 1956). Several investigators have described that the auditory stimuli given to a human body stimulate the hypothalamus and the limbic lobe of cortex in the brain (Klings and Friedel 1933; Leiman 1957; Sakumoto 1957). The higher centers of autonomic nervous system are situated in the hypothalamus and the limbic lobe of the cerebral cortex (Green 1972).

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T. Matoh et al.

Vibration disease induced by the mechanical vibration has been reported to impair not only the functions of the peripheral nervous system and circulatory system and mobility in the upper extremities, but also the function of the central nervous system (Matoh et al., 1974). The main causes of this disease are presumably noises and vibrations generated by a vibrating tool and coldness in the environment. These noises, vibrations and coldness would be regarded as the so-called stresses against a human body. Therefore, it is of interest to know the changes in the function of the autonomic nervous system in patients with vibration disease.

The purpose of the present paper is to clarify the function of the autonomic nervous system in patients with vibration disease as expressed by a digital plethysmography in response to the auditory stimuli.

Subjects and Methods

Fifteen healthy men with averaged age of 43.5±3.1 were selected from 37 of the members of Japanese Self-Defense Force after the examination of blood pressure in order to exclude those with hypertension and after the test of Cornell Medical Index for the purpose of screening of the body condition. Their blood pressures were 123.9±12.5 mmHg in systole and 74.9±8.6 mmHg in diastole. Eighty two male patients with vibration disease had been engaged in forestry as chainsaw operators. Their averaged age was 48.4±7.3. The mean blood pressures were 129.5±12.0 mmHg in systole and 77.5±10.3 mmHg in diastole. The severity of the disease was the third degree according to Andrews-Galantia's criteria (Nakamura, 1971).

The digital plethysmograms and their responses to auditory stimuli were recorded by means of a plethysmograph (Takachio Instrument Co., DC-103) in a sound-proof room. Noises given as auditory stimuli were those recorded from a chainsaw and given by a headphone for 10 sec. The intensity of noises was 90 to 102dB.

The drugs used were tolazoline hydrochloride (Yamanouchi Pharm. Co.), one of the imidazoline derivatives with alpha-adrennergic blocking properties (Caristi, 1972), and sulpiride (Fujisawa Pharm. Co.) which has an inhibitory action against hypothalamus (Fukuda and Takeda, 1969). The chemical structure of sulpiride is shown below.

![Chemical Structure of Sulpiride]

All observations were performed at room temperature (23-24°C).

Results

Digital plethysmographic responses to the auditory stimuli in healthy men and patients with vibration disease

Observations were made on the changes of digital plethysmograms induced...
by auditory stimuli. A typical pattern of the digital plethysmographic response was shown in Fig. 1.

In healthy men the auditory stimuli caused a prompt and marked decrease in amplitude of the plethysmograms. After cessation of the auditory stimuli, the amplitude tended to recover toward the control level within 30 sec (Fig. 1-A). In the patients with vibration disease, on the contrary, there were poor responses to the auditory stimuli (Fig. 1-B). The recovery of the amplitudes was also incomplete, less than 60% of the control value even at 60 sec.

Fig. 2 shows the analysis of the digital plethysmograms taken from 15 healthy men and 82 patients with vibration disease. In the patients with vibration disease the recovery of the amplitude of the plethysmogram ones reduced by the auditory stimuli was retarded as compared with that of the healthy subjects. The time course of the recovery varied considerably from patient to patient.

---

**Fig. 1.** A typical pattern of the digital plethysmogram responded to auditory stimuli in a healthy man and a patient with vibration disease. In a healthy man, there is a good response to the auditory stimuli and the excellent recovery of the reduced amplitudes (A). On the other hand, the response and the recovery are poor in a patient with vibration disease (B). Arrow marks indicate the auditory stimuli for 10 sec.

**Fig. 2.** A diagram of digital plethysmograms taken from 15 of the healthy men and 82 of the patients with vibration disease. o, healthy man (n=15); x, vibration disease (n=82).
Classification of the digital plethysmographic response to the auditory stimuli in the patients with vibration disease

The patterns of plethysmographic responses obtained in 82 patients with vibration disease could be divided into 4 types as shown in Fig. 3: normal (N), intermediate (I), delayed (D) and poor response (P) types. The number of the subjects was 24, 17, 26 and 15 in the types of N, I, D and P, respectively. These types were classified on the basis of the following criteria: In type N, the reduced amplitude elicited by the auditory stimuli recovered to the control value within 30 sec. In type I, the reduced amplitude was recovered more than 80% of the control value at 60 sec. In type D, it was less than 80% of the control value at 60 sec. In type P, the response to the auditory stimuli was poor; the reduction of amplitude

Fig. 3. Typical four types of the digital plethysmographic responses to auditory stimuli in patients with vibration disease. The types of normal (N), intermediate (I), delayed (D) and poor response (P) are shown in A, B, C and D, respectively. The reduced amplitude due to auditory stimuli recovers promptly to the control value in N type (A). In type I, the reduced amplitude is recovered more than 80% of the control value at 60 sec (B). In type D, it is less than 80% (C). In type P, the response to auditory stimuli is poor (D).

Fig. 4. A diagram of 4 types of the digital plethysmograms in the patients with vibration disease. C: N type (n=24); ♦: P type (n=15); +: I type (n=17); ×: D type (n=26).
Digital Plethysmographic Responses to Auditory Stimuli

Table I. Changes of the digital plethysmographic types by the treatments

<table>
<thead>
<tr>
<th>Type</th>
<th>Before</th>
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</tr>
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<tbody>
<tr>
<td>N</td>
<td>N</td>
<td>6/6</td>
<td>100%</td>
</tr>
<tr>
<td>I</td>
<td>N</td>
<td>5/7</td>
<td>71.4%</td>
</tr>
<tr>
<td></td>
<td>I</td>
<td>4/7</td>
<td>57.1%</td>
</tr>
<tr>
<td>D</td>
<td>N</td>
<td>1/12</td>
<td>8.3%</td>
</tr>
<tr>
<td></td>
<td>I</td>
<td>1/12</td>
<td>8.3%</td>
</tr>
<tr>
<td>P</td>
<td>N</td>
<td>3/5</td>
<td>60.0%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>1/9</td>
<td>11.1%</td>
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was less than 30% of the control value. The time courses of responses of four types were illustrated in Fig. 4.

Fifteen healthy subjects revealed type N response in the plethysmograms.

Each type in the patients with vibration disease was shifted to the other types by the treatments, consisting of therapeutic exercises, hot spring cures and drugs of vasodilators. These data were summarized in Table I. Namely, type I altered to type N. Type D altered to type I or N. Type P changed to type I or N.

Mechanism of the digital plethysmographic response to the auditory stimuli in the patient with vibration disease

The experiments were designed to find out the principal cause of the altered response to the auditory stimuli and the delayed recovery of the plethysmogram with a particular interest in knowing its central or peripheral origin.

Fig. 5. A digital plethysmographic response to auditory stimuli in the patient with type D under the influence of alpha blockade. The auditory stimuli decrease the amplitude (1). The intranasal injection of cocaine hydrochloride, 0.5 mg, results in poor response to auditory stimuli (2 to 4). The phenomena at 15, 40 and 90 min after the administration are shown in the figures of 2, 3 and 4, respectively.
A patient with type D was intramuscularly administered with 20 mg of tolazoline hydrochloride. After 50 min, the responses to the auditory stimuli became poor without changes in the time course of the recovery (Fig. 5). This indicates that tolazoline blocks alpha-adrenergic receptors of the blood vessels in the fingers, and that the responses to the auditory stimuli are manifested after the transmission of autonomic nerve impulses to the blood vessels in the fingers.

Intramuscular injection of 50 mg sulpiride, given to the same patient, caused no changes in pletysmographic responses to the auditory stimuli as compared with those of control even after 50 min as shown in Fig. 6. The recovery of the reduced amplitudes, however, tended to become faster. The time course of the recovery became similar to that of type N. This finding clearly suggests that the time course of the recovery after cessation of the auditory stimuli shows the continuity of vasoconstriction of small arteries. In other words, it represents the functional condition of the autonomic nervous center. The condition of the autonomic activity in type D seems to be hyperreactive.

Without tolazoline or sulpiride, there were no significant changes of patterns in the digital pletysmograms responded to the auditory stimuli within at least 60 min.

Relationship between the amplitude of the digital pletysmogram before the auditory stimuli and the digital pletysmographic response to the auditory stimuli

The mean amplitudes of the pletysmograms before the auditory stimuli in 15 healthy men and 82 patients with vibration disease were 4.5±0.9 and 3.8±1.3 mV/V, respectively, the difference being statistically significant (p<0.05). The 82 patients were divided into three groups by the degree of amplitudes before auditory stimuli. Fortyeight patients were included in group B, having the amplitudes...
from 2.4 to 5.1 mV/V. Group A with amplitudes of less than 2.4 mV/V and group C with more than 5.1 mV/V had 10 and 8 patients, respectively. As shown in Fig. 7, there were no significant differences among these three groups. The mean amplitudes of the plethysmograms in types N, I, D and P were 4.1±1.5, 3.7±1.0, 3.9±1.2 and 3.4±1.0 mV/V, respectively. There were no significant differences among them.

**Discussion**

Small arteries in the fingers are innervated with autonomic nerve fibers. The sympathetic vasomotor nerves apparently exert their action on smooth muscle fibers with so-called alpha-receptor sites (Rushmer 1970). The degree of vasoconstriction is directly proportional to the quantity of the nerve impulse of sympathetic nervous system (Yamagishi 1974).

Noises given as auditory stimuli, on the other hand, is known to produce the excitation of the limbic system in the brain (Kluger and Friedel 1933; Lehman 1957; Sakanoto 1957).

As demonstrated in the present study, the digital plethysmographic responses to the auditory stimuli and the time courses of the recovery of the response in patients with vibration disease are different from those in healthy subjects. The reduction of amplitudes induced by auditory stimuli manifests the transmission of autonomic nerve impulses to the blood vessels, and the time course of the recovery of the response shows the continuity of vasoconstriction of arteries. These digital plethysmographic patterns are divided into 4 types. Each type has been shifted from one type to another by the treatments. One of the treatments has been therapeutic exercises. A proper amount of physical exercises results in vagotony. Thus, these types would represent the level of the activity of autonomic nervous system. Namely, types I and D may be hyperreactive, and type P of hyporeactive. In fact, a patient with vibration
disease complains of the disturbances of sound sleep, forgetfulness, the abnormal increase of palmar sweating, the disturbance of circulation and so on (Matoba et al., 1971).

It is concluded that the patients with vibration disease are associated with the disorder of autonomic nervous system. It would be also accepted that the digital plethysmography combined with auditory stimuli is one of the excellent indicators of the level of the activity of autonomic nervous system.

References

SECTION 17
### SUMMARY PAGE FOR

**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s):</th>
<th>Institution and address where research was performed:</th>
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</table>
| C. Ohkubo, K. Miyazaki, T. Genda | Department of Physiological Hygiene  
Tokyo, Japan |

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</table>

**Type & duration of experiment:**

Laboratory - soundproof room  
2 3/4 hours AM, 1 3/4 hours in PM per subject

**Purpose for study:**

To study the effect of intermittent noise on finger pulse amplitude and pulse rate.

**Description of test groups (subjects, sex, etc.):**

- 6 healthy male students 19-24 years old  
- 6 healthy female students 19-23 years old  
- Subjects tested during chair-rest

**Control of other stresses:**

- Laboratory test - constant temp.  
- Background noise - 25 dBA

**Statistical Methods:**

- Factor analysis

**Noise Stimulus:**

- Source: Recorded white noise and train noise by Bruel & Kjaer type 1402 equal spectral characteristics: Low frequency noise, (broad band)-rectangle; train noise, noise level: 65-85 dBA
- Length of exposure: Intermitent - 7 sec. every 5 min. for 2 3/4 hours  
- # of trials: 2 per subject

**CVS Response Measured:**

- Finger pulse amplitude (index finger and middle finger) pulse rate

**Nonaudiitory effects:**

- Pulse rate - slight increase due to noise  
- CVS: "Finger pulse amplitude - decreased markedly due to noise; greater decrease observed due to the white noise; response slower in male subjects  
- Other: annoyance - greater with white noise

**Author's conclusions:**

- Reduced finger pulse amplitude in response to noise is an accurate physiological index of noise effects and indicates noise-induced sympathetic nervous tension. The finger pulse amplitude response is non-specific.

**Evaluation & comments:**

This study confirms some of Gerd Jansen's earlier work.

The effect of train noise and white noise on pulse rate and fingerpulse amplitude was studied in 6 healthy male and 6 healthy female students, 19-24 years old. Each subject was tested during chair-rest in a soundproof room with a background noise level of 25 dBA. The subjects were exposed to recorded intermittent train noise and white noise at varying levels of 65-85 dBA, arranged in random order. Each subject was exposed to noise for 7 seconds every 5 minutes for 1 3/4 hours from 10:30 - 11:15 a.m. and 2:00 - 3:45 p.m. in one day. The white noise had a rectangular pattern (broadband), whereas the train noise had a trapezial pattern, the rise time being 10 dB per 0.08 seconds. Pulse rates and fingerpulse amplitudes were measured during noise and quiet by a photoelectric plethysmograph.

The pulse rates increased significantly due to noise; however, no significant variations were observed with the different noise types or levels. After a latency period of 1-2 seconds, fingerpulse amplitudes were reduced due to noise to an average of 60% of the amplitude during quiet. White noise produced a greater decrease in amplitude than train noise at the same level, possibly due to its broader band. The amplitude response increased with the noise level. The minimum amplitude response was reached later in the male than in the female subjects. The authors conclude that fingerpulse amplitude is a reliable index of the effects of noise, although the response is nonspecific.
RESPONSE OF FINGER PULSE AMPLITUDE TO INTERMITTENT NOISE

BY

Ohiyoji Ohkubo, Kuratoshi Miyazaki and Yasutaka Osada
(Department of Physiological Hygiene, National Institute of
Public Health)

PREFACE

The annoyance aspect of noise is affected by various factors
such as quality, level, duration and rising & falling time of the
noise. Therefore, studies are conducted to make primarily a
psycho-physiological evaluation of the noise exposing combinations
of these factors to subjects who were assigned to judge the
"annoyance" of the noise. On the other hand, however, there
is some room for questioning as to the validity of evaluating
noise based upon the physiological index of the noise effect.
It has been known for a long time that peripheral blood vessels
are induced to contract by the effect of noise. However, it
was Lehmann and Jahnson (1-5) who started to conduct various
experiments using fingertip pulse waves. They investigated
primarily the effect of pure tone and band noise. Therefore,
we have used white noise and train noise as actual noise to
study the effect of noise on fingertip photoelectric pulse waves.

METHODS

Six healthy male students (19-24 years of age, average 21
years old) and six female students (19-23 years of age, average
20.8 years old) were chosen as test subjects. The breakdown of
the students whose present residence was in a quiet, normal and
noisy neighborhood was 5, 5 and 2 respectively. However, the
environmental difference of the subject's residence did not
influence the test results.

The tests were performed in a sound proofed room of this
institute. The background noise of the room was controlled
at approximately 25dB(A), and the room temperature was maintained
at 25 ± 1.5°C. Due to the limited availability of the measuring
instruments, tests were carried out at the rate of 2 subjects
per day. The subjects entered the room at 9.30am, and the
instruments were attached to them. From 10.30 to 12.15, they
were exposed to the noise while they rested on a chair. After
lunch and recess, they came back in the room at one o'clock and
were exposed to the afternoon noise from 2.00 to 3.45pm.
The noise exposed was white noise and train noise. The former was transmitted by an Bruel & Kjaer type 1402 oscillator, and the latter was the noise from the Tokaido line super-express trains passing on the rails built upon raised ground. Both of these noises were recorded on magnetic tape. The recorded changes in the level of the noise exposure are indicated in Figure 1.

![Figure 1](image)

Fig. 1. Paper record of white noise and train noise used in the experiment.

White noise indicated an almost perfect rectangle without any ascending or descending time, while the train noise indicated a trapezoid with an ascending and descending rate of approximately 0.08 sec/10 dB. Both noises indicated a peak level for 7 seconds. In the experiment, these two different noises were exposed at the levels of 65-85 dB(A) by increments of 5 dB. Ten sets of noises were prepared by arranging the types and levels of the noises, and each set of noises was arranged to appear twice in random sampling, and recorded on magnetic tape. The random arranged order of the noises are indicated in Figure 2.

![Figure 2](image)

Fig. 2. Random arrangement of noise exposure.
- Each noise was applied twice within one run of exposure. Noise was exposed every five minutes.

The noise was exposed once in every five minutes allowing an equal interval. The initial signal, 85 dB(A), of the random samples in the figure was white noise, which was placed to adjust the level of the noise exposure and was not the test noise. The level of the exposure was determined in the vicinity of the ears of the subjects. The subjects were exposed to the noise for 7 seconds.
every 5 minutes and allowed to read light literature in between
the noise exposure so that they would not fall asleep. However,
they were instructed to stop reading one minute before the
starting of the noise and not to move their body but sit still.
Further, they were instructed to remain seated and keep the
same posture for approximately one minute after the noise
terminated.

Photoelectric plethysmograph and impedance plethysmograph
of finger-tip pulse waves were taken. The former was taken
by attaching a reflecting type pick-up (MPP-2 model by Nihon
Koden Co.) to the back side of the index finger phalanx of left
hand, and the latter was taken by attaching an electrode (AD3-2
model by Nihon Koden Co.) to the both sides of the middle finger
phalanx of the left hand. Both inputs were drawn on recording
paper through the medium of a DC Amp (AD3-2 model by Nihon Koden
Co.). An example of the record of both plethysmographies are
indicated in Figure 3. However, only photoelectric plethysmographic
records were measured due to various problems relating to the
interpretation of the impedance plethysmography.

Fig. 3. A case of paper record of photoelectric and impedance plethysmographies.
Two subjects were tested in a single experiment.
RESULTS

1. CHANGES IN PULSE RATE

Relative changes in pulse rate were measured from the photoelectric plethysmograph. First, from the paper recordings, the pulse rate of the subjects while sitting still and stabilized during the one minute before the noise exposure was counted, and this rate was designated as an initial value. Next, the pulse waves every 10 seconds including 7 seconds of noise exposure time was read a total of 4 times, and the value which changed most among the 4 was designated as the value after exposure. The relative value(%) of this value to the initial value was calculated. Each subject was exposed to the same noise (for example, 65dB(A) of white noise) a total of 4 times, i.e. two times each in the morning and in the afternoon, and the values obtained at the above 4 readings was averaged and designated as a response value of the subject to that particular noise.

Table 1 indicates the average value obtained by the above described method and the standard error of each 6 subjects according to the types and levels of the noises and the sex of the subjects. The value designated as control indicates the changes in the pulse rate during the period of time one minute before exposure to the noise when the subjects were instructed to remain in a resting position. This value is almost 100%, which indicates that when the subjects obediently followed the above instructions, the pulse rate hardly changed. However, when the subjects were exposed to the noise, the value increased almost all the time to above 100%. Although the rate of increase might have been small, it is obvious that the pulse rate increased by the noise. According to the results of the factor analysis, there was a significant difference between the pulse rate during the noise exposure and the control, there was, however, no significant difference detected among the pulse rates taken under different types and levels of noise.

2. CHANGES IN PULSE WAVE AMPLITUDE

As clearly seen in Figure 3, pulse waves of photoelectric plethysmograph reduced their amplitude as an effect of the noise, which indicates the occurrence of vaso-constriction. Now, designating the average value of the pulse wave amplitude during the stabilized state before the noise exposure as 100%, the relative value of the amplitude immediately before and after the exposure was calculated for each pulse beat and is shown in figure 4.

This diagram is an example of the response of a certain subject to white noise of 80 and 85dB(A). Each beat is drawn in an equal interval, thus the horizontal axis indicating time is not equally spaced. As revealed in figures 3 and 4, the amplitude of the pulse waves after the starting of the noise exposure radically decreased after a 1-2 second latent period, and once it reached
the minimum value it gradually recovered. However, amplitude greatly changed for each beat, so it cannot be necessarily concluded that the minimum value in this diagram indicates the state of amplitude during the minimum blood flow. Consequently, just as done with the pulse rate, the relative values of amplitude at every 10 seconds after the start of exposure were averaged, and the average values and standard errors of the 6 subjects pertaining to its minimum value are shown in Table 2 and in Figure 5(A). In Table 2, the results of the factor analysis are also indicated. The control in the table, same as in the case of the pulse rate, was the value obtained during the time when the subjects were instructed to rest. Even by this instruction alone, amplitude average lowered to 90% indicating a slight vasoconstriction. However, when exposed to actual noise, even a
train noise of 65dB(A), the average amplitude was reduced below 50%, and amplitude reduced radically corresponding to the elevation of the noise level. According to the results of the factor analysis, significant changes are seen in the amplitude of the pulse waves not only by the noise level but also by the type of the noise, and it revealed that pulse amplitude was more affected by the white noise than by the train noise. According to an examination of the differences in amplitudes by the noise levels, among the control; 65-80dB(A); 85dB(A) significant differences were observed.

Table 2. Changes in Amplitude of Pulse Waves

<table>
<thead>
<tr>
<th>Noise level</th>
<th>Male</th>
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<td>n=12</td>
<td>n=6</td>
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<td>n=15</td>
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<td>58±2.3</td>
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<td>80 dBA</td>
<td>53±5.3</td>
<td>53±5.4</td>
<td>53±3.8</td>
<td>49±2.6</td>
<td>44±4.6</td>
<td>42±2.3</td>
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<tr>
<td>85 dBA</td>
<td>46±5.6</td>
<td>45±5.7</td>
<td>44±5.6</td>
<td>42±3.8</td>
<td>43±5.2</td>
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<td>[Control]</td>
<td>30±4.3</td>
<td>30±3.2</td>
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[b] Factor analysis

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<tr>
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<td>2024.4**</td>
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<tr>
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<td>Subject (H8)</td>
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<td>N x S x L</td>
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<tr>
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<tr>
<td>Total</td>
<td>62104.0</td>
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3. TIME TO THE MINIMUM AMPLITUDE

After initiating noise exposure, time required to reach the minimum amplitude was read from the recording paper, and the average values and the standard errors calculated according to the types and levels of the noise are indicated in Figure 5 (A). Also, its factor analytic results are listed in the 2nd column of Table 3. There was no significant difference detected in time required to reach the minimum amplitude regardless of whether the train noise or the white noise was exposed.

![Graph](image)

**Fig. 5.** Changes in photoelectric pulse wave amplitude by noise exposure.
(A) Relative amplitude obtained at the time of maximum vaso-constriction.
(B) Time from the onset of noise taken for the maximum vaso-constriction to appear.
(C) Time from the onset of noise taken for the amplitude to recover to its initial level.

Averages and standard errors for 12 subjects are illustrated.

However, it was greatly affected by the level of the noise, i.e., the time required to reach the minimum amplitude extended corresponding to the increase of the noise level. As for the differences according to the levels, among three groups, 65; 70-75; 80-85dB(A), significant differences were indicated. Significant differences were also detected according to the sex.
Table 3. Factor Analysis of the Changes in Time of Maximum Vaso-constriction (Tm) and of its Recovery (Tr)

(a) Factor analysis (**p<.01, *p<.05)

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<tr>
<td>N×S</td>
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</tr>
<tr>
<td>L×S</td>
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<td>Error</td>
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(b) Significant difference (**p<.01, *p<.05)

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<tr>
<td></td>
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<td>dBA 65 70 75 80</td>
<td>dBA 65 70 75 80</td>
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<tr>
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</table>

Of the subjects, the time required to reach the minimum amplitude was larger in male than in female.

4. TIME TO RECOVERY

The time required for the reduced pulse wave amplitude to recover to its initial value (100%) was measured from the point of time of initiation of the noise exposure, and the average values and standard errors calculated according to the types and levels of noise are indicated in Figure 5(C). The results of the factor analysis are listed in the Tr column of Table 3. This time to recovery was longer when a subject was exposed to the white noise than to the train noise, and further, it extended with the increase of the level of the noise. Among the groups of 55-70; 75-80; 85dB(A) significant differences were observed. However, sex and differences of the subjects did not play an important role for this matter.
The following is a summary of the test results. When exposed to noise, the amplitude of fingertip photoelectric pulse waves critically decreased after one to two second latent period, and it recovered gradually after reaching the minimum value. This response was intensified in proportion to the increase of the levels of noise resulting in the rapid reduction of the amplitude and the extension of the time required for reaching the minimum value and for recovery. The amplitude responded more strongly to the white noise than the train noise although the level of the noises were identical, and the white noise reduced the amplitude and extended the recovery time in great deal. Also, in male subjects, the reduced amplitude took a longer time to recover to its initial level than in female subjects. Pulse rate increased by the effect of the noise, but there was no significant differences caused by the types and levels of the noise in particular. Viewing the above described results, although the changes in pulse rate may not be much, response of the pulse wave amplitude to the noise detected in the tests can be a considerably accurate physiological index of the effect of noise.

As mentioned in the "preface", it was Lehmann and Jansen who conducted many studies using fingertip pulse waves. They first studied then using ballistocardiographs and confirmed that the resistance to the peripheral blood flow was incremented by the effect of noise 1), and then, they continued their studies using a sort of impedance plethysmographies 2) resulting in the achievement of the following convictions: Fingertip pulse waves amplitude decreased by the noise, which was not clearly detectible in infants but manifested better in children of 8-11 years old and presented a normal adult level of decrease in youngers of 19 years old 4) 5); Louder the noise the greater the response became 5); As for frequency, the response of the fingertip pulse waves did not change by the frequency when white noise was exposed but it became greater with the increase of the frequency when a 1/3 octave belt noise was exposed 5); Also, even with the same center frequency, the wider the belt of the noise expanded, i.e., 1/3 octave to one octave to broadband noise, the larger the response became 3). Around the same time as Lehmann and Jansen, Grandjean and his group 6), 7) proved peripheral vaso-constrictions due to noise from the changes in plethysmography and the skin surface temperatures. In comparatively recent years, Fuchs-Schmuck 8) investigated the reduction in the amplitude of fingertip volume pulse waves due to the broadband noise using the same method as Jansen used. Also, Conrad 9) studied the decrease of fingertip pulse wave amplitude based upon photoelectric plethysmographies.

According to the above described data made available up to now, it is quite certain that the noise induces peripheral
vaso-constrictions and thus blood flow reduction-pulse wave amplitude reduction. This response was confirmed at the noise level of 63 dB (A) according to Jansen and his group's data, and 60 or 70 dB (A) according to Fuchs-Schmuck's data. Even in this experiment, as seen in Figure 5(A), 65dB(A) of noise greatly contributed to the response of the subjects compared to the control. Based upon Jansen's data, amplitude was hardly decreased by a 50dB level of noise 5). It is for a future study to find out the lower limit of the potential detection of the response by the plethysmographies applied in this experiment. It was found that the response induced by the white noise was greater than the response by the train noise when the level of both noise was identical, which, for one, may be attributable to what Jansen found, i.e., white noise has a broader belt than train noise and is annoying to the ears. This point was agreeable with the subjective judgement of the test subjects. Another reason for this is assumed to be the abruptness of the occurrence of the white noise, which was manifested in a rectangular shape in the paper record of the noise. It is a future problem to investigate the relationship between the response and the ascending time of the noise. Incidentally, sexual differences affected the time required to reach the minimum amplitude value, and the results of the test indicated that a much longer time was required in male subjects than in female ones. Nevertheless, it is not disclosed yet why it should be so.

Compared to the response manifested in the reduction of the pulse wave amplitude, an increment of the pulse rate was not so ideal an index. Although the pulse rate increased slightly by noise, differences in types and levels of noise did not affect the pulse rate.

Well, peripheral vaso-constrictions and pulse rate increase by noise, the same as salivation reduction and stomach motor inhibition, are the results of a tense sympathetic nervous system caused by noise which worked as a mental and psychosocial stimulus. Lehmann stated 10) that response manifested in fingertip pulse wave amplitude reduction was a response specifically related to noise different from other non-specific tensions of the sympathetic nervous system, which, however, has not been substantiated. Considering that vaso-constrictions occur after a considerably long latent period of 1-2 seconds, and that the constrictrions to a small degree occurred even only by the instructions to remain still, it should be considered that this type of response is a non-specific response which involves mental tensions. However, since the response of fingertip amplitude was detected to vary according to the quality and level of the noise, although it may be a non-specific response, it can be regarded as an accurate index of noise effect. In the future, response to noise in relation to quality, ascending time and duration will be investigated.
SUMMARY

The effect of intermittent noise on the fingertip photoelectric pulse waves was studied. Six male and six female students were chosen and subjected to exposure to train noise and white noise which had a peak level duration of 7 seconds once every 5 minutes for 105 minutes twice a day, once in the morning and the other in the afternoon. Timing of the train noise indicated a trapezoid with approximately 0.08sec/10dB for both the ascending and descending noise while the timing of the white noise indicated a rectangle. Peak level was exposed to the subjects in 5 phases from 65-95dB(A) measured at the ears using increments of 5dB. Each noise was exposed in random order.

Pulse rate slightly increased by the effect of the noise but was not detected to be influenced by the differences in the types and levels of the noise.

The pulse wave amplitude taken by photoelectric plethysmography decreased radically after 1 to 2 seconds of the latent period subsequent to the commencement of the noise exposure. The rate of decrease grew larger proportionately to the increase of the exposure level, and also the white noise affected the amplitude more than the train noise. The time required to reach the minimum amplitude became longer with the higher levels of the noise but the differences in the types of noise did not affect the time. However, it took longer in males than in females. The time required to recover to the initial level was also found to be influenced more by white noise than by the train noise. The response to white noise which was stronger than the train noise may be due to the broadness of the band and the abruptness of the occurrence of the noise, which coincided with the "annoyance" that the subjects complained about concerning the white noise.

The reduction of the fingertip pulse wave amplitude is responsible for the non-specific tension of the sympathetic nervous system. Based upon the results of the tests described above, this response to the noise can be considered a quite accurate physiological index of noise effect.

REFERENCE MATERIAL


Response of Finger Pulse Amplitude to Intermittent Noise

Chiyoji Okihudo, Kurtoshi, Miyazaki, and Yasutaka Osada
(from the Department of Physiological Hygiene, the Institute of Public Health, Tokyo)

C. Okihudo, K. Miyazaki, and Y. Osada Responses of finger pulse amplitude to intermittent noise. Bull. Inst. Publ. Health, 25 (1): 1-8, 1978.—Six male and six female students were subjected in this study and two of them were tested in a single experiment. They were sitting in a sound-proofed and air-conditioned room and were exposed to white noise (W.N.) or train noise (T.N.) every five minutes for 10 minutes. W.N. and T.N. were reproduced through tape-recorder and speaker at peak levels of 85-85 dB (A) at the ears of the subjects. Duration of peak level of noise was fixed for 7 seconds (Fig. 1). The exposure was so arranged that each noise appeared twice but in random order (Fig. 2). The experiment was performed twice a day, in the morning and afternoon, under the same schedule and condition.

Finger pulse waves were recorded by photoelectric inductor attached at the index finger phalanx of left hand and their amplitude and rate were measured (Fig. 3). Impedance plethysmograph was also taken from the middle finger of the same hand but was not served for measurement because of some uncertainties in physiological explanation of its amplitude changes.

Pulse rate counted from paper record of pulse waves slightly increased by noise but there was no significant difference of the increment according to type and level of noise (Table 1).

Amplitude of pulse wave was remarkably decreased by noise with 1 to 2 seconds latency and recovered gradually to its initial level. This response indicated peripheral vaso-constriction at finger induced by noise. For quantitative analysis of the response, changes in pulse wave amplitude were expressed in percentages of its initial value (Fig. 4). Minimum value of relative amplitude thus obtained became smaller according as the noise level increased. W.N. produced smaller values than T.N. did, even when the noise level was identical (Table 2 and Fig. 4). The time taken for the pulse wave amplitude to reach to its maximum constriction (Tm) and to return to its initial level (Tr) was measured from the onset of noise exposure. The higher the noise level, the more both of Tm and Tr were elongated (Table 3 and Fig. 4). W.N. elongated Tr more than T.N. did, while such difference was not observed with Tm. Sexual difference was noticed only with Tm: longer in males than in females, but the reason was not clear. Above findings indicated that the higher the level of noise, the larger the vaso-constrictor response. W.N. had a more potent effect than T.N. because of its broadband characteristics.
and of promptness of occurrence. Thus, the response of finger pulse wave amplitude was concluded to be sensitive enough to be used as a psychophysiological index of noise effect.

### Method

Patients were divided into three groups: 6 male subjects (19-24 yrs, mean 21 yrs) and 6 female subjects (19-23 yrs, mean 20.3 yrs). All subjects were free from any known disease and were in good health. The experiment was conducted on a single session.

### Experimental Procedure

- **Subjects:** Male and female subjects were divided into three groups.
- **Stimuli:** White noise and tone pips were used as stimuli.
- **Procedure:** Each subject was seated in a sound-attenuated booth.
- **Response:** Finger pulse wave amplitude was measured.

### Results

Fig. 1 shows the paper record of white noise and train noise used in the experiment.

**Figure 1:** Paper record of white noise and train noise used in the experiment.

**Figure 2:** Random arrangement of noise exposure.

Each noise was applied twice within one run of exposure. Noise was exposed every five minutes.
Fig. 3. A case of paper record of photoelectric and impedance plethysmography.
Two subjects were tested in a single experiment.

結果

1. 噪音数の変化

Fig. 3 に示した光電プレシスメーターから、脈拍数の相対的变化を計測した。まず正規化前、脈拍数の相対的変化を示した。脈拍数が正常を示す。脈拍数の相対的変化を示した。脈拍数が正常を示す。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正常を示した。脈拍数が正
Table 1. Changes in Pulse Rate by Noise Exposure

<table>
<thead>
<tr>
<th>Noise level</th>
<th>Shinkansen-train noise</th>
<th>White noise</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>65 dBA</td>
<td>103±1.3</td>
<td>103±1.2</td>
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<td>70 dBA</td>
<td>103±0.5</td>
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<td>80 dBA</td>
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<td>85 dBA</td>
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<td>102±0.8</td>
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<tr>
<td>Control</td>
<td>100±1.3</td>
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</tr>
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[b] Factor analysis

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<th>df</th>
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[c] Significant difference (**p<.01, *p<.05)

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<td>70</td>
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4 障害気味と間欠騒音

bull, inst. publ. health, 23 (1): 1070
Fig. 4. Two cases of relative changes in finger pulse wave amplitude. Relative amplitude is expressed in percentage of its initial value. In this graph, interval between pulses is fixed and thus time scale is not equally spaced.

Table 2. Changes in Amplitude of Pulse Waves

(a) Percent of initial value ± S.E.

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<tr>
<th>Noise level</th>
<th>Male</th>
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<th>Total</th>
<th>Male</th>
<th>Female</th>
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<td>48±4.0</td>
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<td>70 dBA</td>
<td>51±3.6</td>
<td>52±4.0</td>
<td>52±4.0</td>
<td>43±4.1</td>
<td>45±3.5</td>
<td>44±2.6</td>
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<tr>
<td>80 dBA</td>
<td>50±3.4</td>
<td>50±2.7</td>
<td>50±3.7</td>
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<td>42±3.1</td>
<td>47±2.6</td>
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<tr>
<td>90 dBA</td>
<td>53±4.5</td>
<td>53±3.4</td>
<td>53±3.8</td>
<td>40±2.6</td>
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<td>42±2.6</td>
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<tr>
<td>Control</td>
<td>53±3.4</td>
<td>52±3.2</td>
<td>52±3.6</td>
<td>38±3.6</td>
<td>43±3.2</td>
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(b) Factor analysis

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<td>Level of noise [L]</td>
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<td>L x S</td>
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<td>N x S x L</td>
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Table 3. Factor Analysis of the Changes in Time of Maximal Vaso-constriction (Tm) and of Its Recovery (Tr)

(a) Factor analysis (**p<.01, *p<.05)

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<th>F(Tr)</th>
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<td>Level of noise [L]</td>
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<td>Subject [S(S)]</td>
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<td>N x S</td>
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</tr>
<tr>
<td>L x S</td>
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<tr>
<td>N x L x S</td>
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<tr>
<td>Error</td>
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(b) Significant difference (**p<.01, *p<.05)

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<table>
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されると、発赤の光電顕微鏡の幅は 1～2 毛の間隔を経て急激に減少し、最 小値に達したあと長時間に回復す る。発赤のレベルが高いほど、この反応が強く、発赤 極が著しく、発赤開始時間と分泌両差は長くなる。列腺組織より白色伝えの方が同じレベルであっても反応が短く、腺伝導と回復時間延長が著しい、ま た最大到達時間を女子より男子の方が早い。腺伝導 は興奮によって増加するが、興奮の役割やレベルによ る差は有意でなかった。以上の結果からみると、腺伝導 数の変化はともかわらず、腺伝導減少反応はかなり明瞭な

生理的示現であるといえる。
「まえがき」へのたたきように、指示の発表を用いて多くの研究をおこなったのは Lehmann や Jansen であった。彼らは発表、パルスストラディオグラフを用いて研究し、発表によって第二血流脈拍が増大することを確認したほか、ついて第1回のインピーダンスブレッサーコープを発表し、発表の脈拍の振幅が発表によって増大すること、この発表は発表であって意識をなく、5〜11才で充分反応するようになり、10才ごろ成人なみの反応に達することを示し、と大いにほとんど反応が大であること。発表は発表、パルスストラディオグラフ発表者では発表数の高いほど反応が大きく、または同じ発表数を持っているように、発表、パルスストラディオグラフ、発表、発表者と発表者が hôるについて反応が大きくなっている。発表、Grandjean らによればパルスストラディオグラフと発表数発表の関係から、発表による血流量脈拍の変化を示した。発表の脈拍数においては Spence-Schmuck らは発表と発表方法で発表脈拍数を発表する発表脈拍数の脈拍を減少させし、また Conned らは発表パルスストラディオグラフによって発表脈拍数脈拍の減少を示している。発表脈拍数脈拍の反応を示すと、発表増加脈拍数は良い

【A】
【B】
【C】

Fig. 5. Changes in photoelectric pulse wave amplitude by noise exposure. (A) Relative amplitude obtained at the time of maximum vaso-constriction. (B) Time from the onset of noise taken for the maximum vaso-constriction to appear. (C) Time from the onset of noise taken for the amplitude to recover to its initial level. Averages and standard errors for 12 subjects are illustrated.

前述の光電激波と呼ばれる過度の視覚の影響についてある。例として、男性および女性、成人の平均的な光学刺激を用いる場合、10秒間の強度の光を観察すると、視覚の反応が異なることが示された。特に、女性の方が男性よりも反応が弱い傾向が見られた。この結果は、視覚の反応が性差により異なる可能性を示唆している。
<table>
<thead>
<tr>
<th>STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)</th>
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<tbody>
<tr>
<td>Principal Investigator(s):</td>
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<tr>
<td>Daryush Parvizpoor M.D., D.I.H.</td>
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<tr>
<td>Institution and address where research was performed:</td>
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<tr>
<td>Dept. of Occupational Health</td>
</tr>
<tr>
<td>University of Tehran, School of Public Health</td>
</tr>
<tr>
<td>Tehran, Iran</td>
</tr>
<tr>
<td>Investigator's Phone No.</td>
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<tr>
<td>Sponsoring Organization:</td>
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<td>University of Tehran, Iranian government</td>
</tr>
<tr>
<td>Citation:</td>
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<tr>
<td>Parvizpoor, D. Noise exposure and prevalence of high blood</td>
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<td>pressure among weavers in Iran.</td>
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<tr>
<td>Type &amp; duration of experiment:</td>
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<tr>
<td>Epidemiological survey of occupational exposure</td>
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<tr>
<td>to noise in three textile mills in Iran.</td>
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<tr>
<td>duration of study - not given</td>
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<td>Purpose for study:</td>
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<tr>
<td>To see if a greater percentage of people</td>
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<tr>
<td>occupationally exposed to high noise levels</td>
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<tr>
<td>were hypertensive compared to similar controls</td>
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<tr>
<td>of same age.</td>
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<tr>
<td>Description of test groups (subjects, A, B, etc.):</td>
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<tr>
<td>Test group: 821 male weavers (aged 19-59, with 9-20 years</td>
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<td>in the mills) in three textile mills in Iran.</td>
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<tr>
<td>Control group: 412 people (aged 19-59, unspecified length of</td>
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<td>employment) of similar socio-economic backgrounds who</td>
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<td>worked in light industries with no high occupational</td>
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<td>exposure to noise (selected at random from same geographic</td>
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<tr>
<td>area).</td>
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<td>Control of other stresses:</td>
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<tr>
<td>Other stresses besides noise included cotton dust (7.8 mg/H),</td>
</tr>
<tr>
<td>heat, high humidity.</td>
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<td>Statistical Methods:</td>
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<tr>
<td>Methods not reported.</td>
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<tr>
<td>difference between tests &amp; controls significant of 5% (0.01)</td>
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<td>Noise Stimulus:</td>
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<td>source: machinery in textile mill</td>
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<td>noise level: 96 dBA average</td>
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<tr>
<td>length of exposure: daily for 9 - 20 yr. (duration of</td>
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<tr>
<td>employment)</td>
</tr>
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<td>1 of criteria: not applicable</td>
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<td>Author's conclusions:</td>
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<tr>
<td>A higher percentage (statistically significant) of males</td>
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<td>occupationally exposed to noise were hypertensive than in the</td>
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<td>control group. Hypertension also occurred at a</td>
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<td>younger age in the 96 dBA noise-exposed group. There</td>
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<td>appeared to be a direct correlation between</td>
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<td>length of exposure to 96 dBA noise and hypertension.</td>
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<td>Evaluation &amp; comments:</td>
</tr>
<tr>
<td>1. The blood pressures were taken only once per subject.</td>
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<tr>
<td>2. The large number of subjects and generally good design of</td>
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<td>study compensate for these points.</td>
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The occurrence of high blood pressure was studied in 821 male weavers in three textile mills in Iran. The average noise level in the mills was 96 dBA, with no major variations. The weavers in the study were screened with a medical exam and a questionnaire to exclude those with family histories of cardiovascular, renal, or thyroid diseases. The blood pressures of the weavers were compared to those of a randomly selected control group of 412 people from the same geographic area and from similar socioeconomic backgrounds. The subjects in the control group were employed in light industries, with no occupational exposure to high noise levels. Blood pressure readings were taken after 5-10 minutes rest under quiet conditions before work in the morning. Tables are included which show the percentage of subjects who are hypertensive (160/95 mm mercury or more), normotensive (below 140/90 mm mercury), and borderline by age and length of employment. There was a moderate increase in the prevalence of hypertension with age in the control group. The increase was greater in the weavers occupationally exposed to 96 dBA, and hypertension occurred in younger age groups. The hypertension rates for each age group were as follows:

<table>
<thead>
<tr>
<th>Age</th>
<th>Weavers</th>
<th>Controls</th>
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<tr>
<td>20-29</td>
<td>1%</td>
<td>0%</td>
</tr>
<tr>
<td>30-39</td>
<td>7.3%</td>
<td>1.2%</td>
</tr>
<tr>
<td>40-49</td>
<td>12.1%</td>
<td>6.5%</td>
</tr>
<tr>
<td>50-59</td>
<td>27.1%</td>
<td>8.6%</td>
</tr>
<tr>
<td>Total</td>
<td>8.5%</td>
<td>2.4%</td>
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</table>

A higher percentage of hypertension was associated with longer occupational exposure to high noise levels—26.1% of the weavers with over 20 years experience were hypertensive. The study shows that occupational noise levels above 90 dBA can produce nonauditory physiological effects. Other stressors in textile mills that could contribute to hypertension include cotton dust, high temperature and humidity, and other adverse environmental conditions.
Noise Exposure and Prevalence of High Blood Pressure Among Weavers in Iran

Daryoush Parvazpoor, M.D., D.I.H.

651 weavers working in three textile mills were examined in this survey. It was found that 0.5% of the workers were hypertensive and 12.4% borderline. All these rates are significantly different from those of the control groups. Also there is a clear increase in the prevalence of hypertension and borderline cases in the weavers in relation to the length of employment. These findings emphasize the need for, at least annually, periodic examination of the entire work population in order to detect early such adverse health effects and to consider suitable medical care.

Several studies, carried out on animals and man, have described nonauditory effects of noise. In animals: Medoff and Bongiorni\(^1\) provided objective evidence on the influence of audiogenic stimulation on systolic blood pressure by exposing rats to a constant air blast for 5 to 10 minutes per day for several weeks. McCann et al\(^2\) demonstrated, by adrenalectomy in noise induced hypertensive rats, that the adrenal cortex mediated the elevation of blood pressure occurring during prolonged auditory stimulation. Smit\(^3\) reported that in rats exposed to a long period of intense noise stimulation the rise in blood pressure persisted for at least seven months after the audiogenic stimulation ended.

In man, under experimental conditions: Anguel\(^4\) studied 26 men exposed to pure sounds of 125, 1,000, 5,000 and 10,000 cps and intensities between 65 and 93 dB for 1-hour periods, and observed substantial elevations in the free plasma 17-OHCS and urinary 17-Ketogenic steroids. Fuchs and Vogel\(^5\) examined pulse volume, blood pressure and pulse frequency in 13 subjects exposed to noise of 59, 65.75 and 85 dB for 10 minutes and observed significant vasocostrictory effects, decreased systolic and increased diastolic blood pressure and tachycardia.

In field studies: Meinhard and Monier\(^6\) detected a significantly higher frequency of cardiovascular abnormalities, including hypertension, in noise injured persons in the District of Halle as compared to the cardiovascular morbidity of the general population of the same area. Kavousi\(^7\) reported that among people occupationally exposed to noise of about 115 dB at a kilo in Tehran, the prevalence of high blood pressure increased considerably with length of employment independently of the prevalence increase due to age.

These data indicate adverse health effects of noise occurring in other systems besides the auditory one. In order to investigate the effect of long-term occupational exposure to noise on blood pressure in a generally steady population, a study was carried out in industrial plants providing such conditions of exposure in the Isfahan area of Iran. This report presents the findings of that investigation.

Materials and Methods

Three textile mills were randomly chosen from the 20 mills with more than 200 employees in the area. 621 weavers were selected for this study after excluding, by means of a questionnaire and medical examination, seven employees with a family history of hypertension, hyperthyroidism, renal and cardiovascular diseases. As the number of female workers in the investigated plants was very low, the study was carried out on males only.

The average noise level at the three textile mills was 96 dBA without significant variations and the cotton dust concentration in the workshops' air was about 7.0 mg/m\(^3\).

Blood pressure was measured on the right arm in the sitting position using the spring sphygmomanometer, after 5 to 10 minutes rest in a quiet room before beginning the day's work. The blood pressure findings were classified according to the recommendations of the WHO Expert Committee\(^8\) into three groups: (a) Normotensive: Systolic blood pressure below 140 mm Hg and diastolic blood pressure below 90 mm Hg. (b) Hypertensive: Systolic blood pressure 150 mm Hg or more or diastolic blood pressure 95 mm Hg or more. (c) Borderline: In between these figures.

A control group of 412 persons of similar socio-economic conditions as the weavers who were working in light industries without occupational exposure to intensive noise were randomly selected from the population of the same area.

Findings

Table 1 shows the distribution of the 412 persons in the control group by age and blood pressure level. Ninety three percent were considered to be normotensive, 2.4% hypertensive and 4.6% borderline. There is a significant association between age and hypertension starting at the 40-49 age group.

Table 2 shows the distribution of the 821 weavers by age and blood pressure level. Only 79.1% of them were considered to be normotensive, while 8.5% were hypertensive and 12.4% borderline. All these rates are significantly different from those of the control group (\(p < 0.01\)). An association between age and hypertension may also be observed among the weavers but the significant increase in the hypertension rates starts at a lower age group (30-39) as compared to the control group (40-49).

Since the three textile mills studied have a very low turnover of workers, length of employment and therefore noise exposure are closely related to age. Table 3 shows the distribution of the 821 weavers by length of employment and blood pressure level. A clear increase in prevalence of hypertension and borderline cases may be observed in relation to length of employment.
Discussion

The findings for the control group do not differ significantly from those described by Nadivi et al who reported that hypertension is becoming a frequent condition in rural areas of Iran.

The findings among those men employed at the textile mills show that they have a significantly greater risk of developing hypertension and that this difference appears already at relatively young ages (20-29) and increases with length of employment. In many industrial activities workers are exposed to much higher noise levels than the 96 dBA observed at the textile mills studied. The findings reported suggest that occupational exposure to noise that slightly exceeds the permissible levels of 85-90 dBA does produce nonauditory effects. In this respect it should be mentioned that Martin et al reported an increased risk of noise-induced hearing loss at noise levels between 85-90 dBA, from 4.0% to 23.5% above the 10% normally impaired due to presbycusis, for those subjects 50 to 65-years of age.

This last fact suggests that the people investigated in this survey should again be examined, both for assessing the development of the nonauditory effects as well as their hearing capacity.

Since other pathogenic factors, besides noise, such as high temperature, high humidity, cotton dust, etc. may play a role in the etiology of high blood pressure among weavers, further studies in plants where the environmental conditions, rather than noise, are different are required to learn more about the long-term noise effects on the blood pressure of occupationally exposed people.

The data reported are in keeping with Kavousi's findings regarding the harmful effects of noise on blood pressure in occupationally exposed people and call for provision of suitable protective devices against noise exposure.

Conclusions

1. People not exposed to intensive noise in their working environment in the Isfahan area showed a moderate increase in hypertension prevalence with age: up to 29 years, 0%, 30-39 years, 1.2%, 40-49 years, 6.5% and 50-59 years, 8.6%.

2. Textile mill workers of the same area, exposed to an average noise level of 96 dBA, showed in comparison with the control group, an increase in hypertension prevalence with age, characterized by its occurrence in younger age groups and at higher rates: Up to 19 years, 0%, 20-29 years, 1%, 30-39 years, 7.3%, 40-49 years, 12.1% and 50-59 years, 27.1%.

3. The fact that 26.1% of the weavers with more than 20 years of exposure are hypertensive, suggests a direct association between length of exposure to a noise level of about 96 dBA and hypertension. However, the fact that only 24.1% of them are hypertensive also suggests a particular role played by individual reactivity.

4. These findings emphasize the need for, at least annually, periodic examinations of the entire work population in order to detect early such adverse health effects and to consider suitable medical care.

5. A minimum preventive programme will consist of providing protective devices, educating the people regarding the need for them and supervision in their use throughout working hours. This could provide a reasonable basis for assessing, in prospective studies, the efficiency of such protective devices in preventing adverse health effects of industrial noise.

References


SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

Principal Investigator(s):
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Investigator's Phone No. Sponsoring Organization
(305) 474-6435 University of Miami School of Medicine
EPA/OMAC

Citation:

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<th>θ of Ref. 's</th>
<th>θ of Fig. 's</th>
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<tr>
<td>104</td>
<td>9</td>
<td>English</td>
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</table>

Type & duration of experiment:
in acoustically isolated booths - 3 experiments
(1) 60 days—signalled noise bursts
(2) 66 days (36 d. quiet & 30 d. noise)
(3) 190 days—quiet

Purpose for study:
to study the cardiovascular effects of noise in a species closely related to man under controlled conditions

Description of test groups:
SD: 180 C57/BL6, saline
3-4 kg. female Rhesus monkeys: 1 for each of the 3 experiments. Each monkey had an implanted blood-pressure catheter in the femoral artery.

Control of other stressors:
controlled laboratory (effect of chair restraint studied alone)

Statistical Methods:
not specified

Noise Stimulus:
(1) source: speaker (2) recorded urban noise
(3) none

Spectral characteristics: spectra given
(1) broadband
noise level: (1) 112 dBA; (2) 78 dBA
(3) no noise—chair restraint only.

Length of exposure:
(1) 9 sec.
(2) 12 hr. /day-30 days (3) none
θ of trials:
(1) 6 per day-30 days
(2) 1 continuous for 12 hrs. daily (3) none

CVS Response Measured:
heart rate
blood pressure (BP)

Nonauditory effects:
(1) both heart rate and BP increased due to CVS noise; baseline BP increased by an average of 28%, significantly
(2) major increase in heart rate and BP in 1st few hrs. (3), (4) of time in a 1 hr. before (5) (2) no sustained changes in heart rate
Other behavioral activity

Author's conclusions:
Unexpected (intermittent) noise and continuous noise can cause sustained cardiovascular changes, which can become independent of a noise stimulus. Noise may be a contribution to the long-term development of cardiovascular disease.

Evaluation & comments:
The use of only 1 monkey per experiment is a weak point—individual variations in responses were not studied.

Three experiments on the effects of noise on heart rate and blood pressure in Rhesus monkeys are reported. A different 4 kg. female with a blood pressure catheter implanted in the femoral artery, served as the subject for each experiment. The monkeys were placed in restraining chairs in double-walled acoustically isolated booths for the duration of the studies. The first experiment used a classical conditioning procedure with a red light as the conditioned stimulus (CS) and 112 dBA broadband noise at the monkey's ear as the unconditioned stimulus (UCS). The most common schedule was as follows: the CS was presented first for 6 sec., then the UCS (noise) for 9 sec. Baseline values were recorded for 10-15 sec. before the CS. The conditioning trials were presented up to 8 times per day for 30 days. During the final 30 day period, the stimulus schedule was held constant. Evidence of some adaptation to the noise stimulus was obtained, since during the final 12 days of the experiment, the magnitude of the responses (heart rate and blood pressure increases) decreased. An apparently conflicting finding was an overall average increase in the baseline blood pressure of 28% over the 30 day interval, which is not an adaptive response. The authors explain the apparent contradiction in terms of two different mechanisms operating: a neurological short-term reaction (adaptation) and a hormonal long-term reaction (upward shift in baseline level). The second experiment consisted of a 30 day control period and then a 30 day test period, during which the monkey was exposed to noise continuously for 12 hours per day. The noise stimulus was a recording of urban traffic noise with an average equivalent noise level (Leq) of 78 dBA. Major increases in heart rate and blood pressure due to noise occurred in the early morning hours and declined throughout the day for the 30 day period. The blood pressure increase began 1 hour before the noise stimulus was turned on, possibly an anticipatory response. The third experiment examined the effect of chair restraint alone on a monkey for 190 days. No sustained effect on heart rate and blood pressure was observed. The authors conclude that both intermittent and continuous noise stimuli can cause sustained changes in cardiovascular responses, which can be independent of the noise. Noise may contribute to the development of cardiovascular diseases in humans.
NOISE AND CARDIOVASCULAR FUNCTION IN RHESUS MONKEYS

E. A. PETENSON, J. S. AUGENSTEIN, B. S. HOSEK, K. I. KLOSE, R. MANAS, I. BLOOM, S. LOVETT, and D. A. GREENBERG
Division of Auditory Research, Department of Otolaryngology
University of Miami School of Medicine, Florida

I. INTRODUCTION

For the foreseeable future, urban societies will continue to be noisy. (1) Divisive and pervasive activities, including those associated with transportation, (2,3) work, (4) and even leisure (5–10) contribute to the contemporary din. It has been estimated that some seventy million people in this country alone are exposed to average noise levels greater than 60 dbA, and six hundred thousand more are exposed to average noise levels greater than 80 dbA. (11) It is not surprising, therefore, that in recent years the question of whether or not such levels eventually become injurious to health has developed into an important scientific and medical issue which has been recognized not only by scientists and physicians, (12–16) but by public administrators (17) and private citizens as well, (18)

Much work has been published which bears on the relationship between noise and various neurological, hormonal and behavioral responses in both animals and humans. (13, 19–22)

Acute responses to sudden noise are well-known and were summarized some time ago by Rosen in the following way: "...the blood vessels constrict, the skin paler, the pupils dilate, the eyes close, one winces, holds one’s breath and the voluntary and involuntary muscles tense. Adrenalin is suddenly injected into the blood stream which increases neuromuscular tension, nervousness, irritability and anxiety." (22)

In addition to producing acoustic trauma, prolonged exposure to noise is claimed to foster a wide range of non-auditory physiological changes, such as: increased adrenal cortical activity (24, 25) with associated complicated changes in hormone balance (26, 27, 28) and blood-urea composition, (29–32) gastric ulcersation, (33, 34) adrenal hypertrophy, (34) thymus hypoplasia, (32) sexual-reproductive, developmental and neurological anomalies, (21, 35, 36) decreased resistance to viral diseases, (37) decreased immunological reactivity, (38) decreased fetal catecholamine, (39) increased cholesterol deposition and atherosclerosis (31) and increased susceptibility to chemically-induced cancer. (40)

1A portion of this report was read before a Joint Acoustical Society of America—Ilexiles '76 Technical Session, "Noise: Its Significance for the Individual and Society," on April 7, 1976, Washington, D.C.

2Department of Neurological Surgery

3Department of Psychology
Changes in salivary gland secretions, (41) blood sedimentation rate, (42) blood-brain barrier permeability, (43) cerebral flow, (44) transketolase enzyme activity, (45) brain metabolism (46) and function, (47) respiration, (48) and the kidneys (49) have also been noted.

Many of these changes are said to be protective reactions associated with the development of the classic General Adaptation Syndrome. (50)

Because of the obvious implications for health related to malfunctioning of the heart and blood vessels, the cardiovascular system has been singled out for extensive study and numerous experiments have been performed under a variety of noise exposure conditions.

Cardiovascular studies can be conveniently classified as those in which animals are used as models for understanding human reaction to noise and those in which humans are used. Human studies can further be divided into those which are carried out under field conditions or retrospectively and those which are carried out under laboratory conditions.

Animal studies originating in this country predominantly have used rodents and lagomorphs. (51, 52) In the typical paradigm, rats or rabbits are subjected to intense noises of varying character and duration. Small to significant increases in blood pressure and heart rate, as well as certain changes in heart structure have been reported. (53, 54)

Studies in which these species are used to specify noise effects have been soundly criticized. (12, 13, 55) The most commonly voiced complaint centers about the special susceptibility of numerous rodent species to noise-induced convulsive behavior. (56, 57) Another distinctive reaction of rats is the increase in blood pressure associated with moving these animals to unusually quiet surroundings. This phenomenon has recently been labeled "sound withdrawal hypertension." (58, 59) By contrast, blood pressure in humans goes down under conditions of acoustic isolation. (60, 61) Also, rodents and rabbits often respond to stressful stimuli, including noise, with immobility or freezing. The physiology underlying such behavior, particularly the cardiovascular aspects, differs importantly from that associated with the classic "fight or flight" behavior (62) prominent in anthropoid species.

A final criticism relates to selection of inappropriate stimulus parameters. Firstly, the sound pressure levels used are usually well above present or even projected environmental values, and secondly, the major energy of these intense stimuli often lies below the range of frequencies to which rats, at least, are most sensitive. (63)

In short, the value of research in which acoustically aberrant species are bombarded with unrealistically intense "infra-sound" in order to infer human reactions to lifelong noise exposure seems slight indeed.

In human studies based on field data, changes in cardiovascular function have been noted mainly in the industrial setting because, despite the fact that urban noise levels have generally been increasing over the years, still higher levels are to be found in the work place. (64-74) Many investigators in eastern and western Europe and Russia have noted the relation between cardiovascular anomalies
and noise exposure occurring during work hours. These studies can generally be described as retrospective in the sense that noise-induced changes are not traced as they occur but are reviewed after the workers have been exposed for a number of years.

In this form of research, factors other than noise cannot be ruled out as causative agents of the effects noted. Nevertheless, there does appear to be a consensus that the noisier the industrial environment within which a worker must perform, the more likely he is to develop cardiovascular diseases, including waveform disorders, peripheral circulatory disorders and hypertension.

The criticism most frequently leveled against the bulk of field-industrial studies is that the often slight differences in cardiovascular function between exposed and non-exposed workers may be accounted for by factors not directly associated with noise. These include the anxiety of working with dangerous machines under conditions of poor ventilation and excessive heat and vibration, as well as problems associated with job security and personnel selection. A second major criticism relates to stimulus control. Off-the-job noise exposure has generally not been accounted for and thus, not controlled. Indeed, even on-the-job exposure has not yet been precisely specified because of the continuously changing relationship between the worker and various noise sources. Because of these shortcomings, the results of field-industrial studies have to be considered at this time provocative rather than definitive.

Better control of stimulus-response parameters is possible in the laboratory setting. Work by Jensen and his colleagues over the past 15 years has provided many of the details of human cardiovascular adjustment to sounds. (70–72) Included among their many significant findings is the observation that little adaptation of peripheral vasoconstriction responses to noise occurs with repeated testing over a span of 6 mo to 3 yrs. Other, mainly short term, laboratory studies (75–80) have, with one exception, (81) noted changes in heart rate, blood flow and blood pressure resulting from exposure to noises ranging from "muffled rustling" to sonic booms.

Most laboratory studies performed to date can be legitimately criticized for their failure to control extra-experimental noise exposure and for their short duration.

II. EXPERIMENTATION

We have undertaken a long-term study of the relationship between noise and cardiovascular function in our laboratory which is designed to overcome, at least partially, the deficiencies inherent in much of the previous work: by studying cardiovascular responses in a species closely related to man and by maintaining rigorous and continuous control of the acoustic environment of the subject. Work described below represents the first three studies carried out under this program.

EXPERIMENT I

In our first experiment, we attempted to trace the course of cardiovascular responses to noise over an extended period. We made no attempt to emulate life-like stimulus conditions.
Methods and Procedures.

Our subject was a young, female Rhesus monkey of about 4 kg weight. A blood pressure catheter was surgically implanted in the abdominal aorta via the femoral artery. During recovery, the monkey was continuously restrained in a specially designed chair. It remained in this chair for the duration of the experiment and, in keeping with previous observations, it did so without apparent ill effects. The blood pressure catheter was continuously infused with a saline and heparin solution in order to prevent clotting and to maintain catheter patency.

The restraining chair and auxiliary equipment were placed in a double-walled, acoustically isolated booth. Responses measured included behavioral activity, blood pressure and heart rate. Signals from the appropriate transducers located within the booth were amplified and fed into a polygraph which provided a graphic record of the raw data.

In this experiment, a classic conditioning procedure was used. A broad band noise measuring 112 dB at the monkey's ear served as the unconditioned stimulus (UCS). It was obtained by driving a speaker with square wave pulses at a rate and pulse duration which by trial and error had proven to be most disruptive to the ongoing behavior of this particular animal. A narrow band analysis of the UCS is shown in Fig. 1. Major energy extended from about 0.4–4 kHz.

In order to distinguish differences which might occur in the learned and unlearned responses associated with this potentially stressful stimulus, a signal for the noise (CS) was also used in most of the trials. CS was a red light placed well within the animal's field of vision.

Fig. 1. Narrow band (10–v) analysis of noise stimulus used in Exper. 1. "O" dB equals 112 dB. Only peaks are shown. Frequency in kHz.
Various conditioning trials were presented up to 8 times/day. A baseline (control level) for heart rate and blood pressure was recorded for 10 to 15 sec immediately prior to CS, UCS, or CS–UCS presentations. In the most common schedule, the CS was presented first for 6 sec and then the UCS was presented for 9 sec directly upon termination of the CS. Responses were monitored during CS and UCS presentation and for 5 min after termination of the UCS.

Results.

Heart Rate, Acute Responses: Over the final 30-day interval of this experiment, during which time stimulus conditions were held constant, a slight but consistent decrease in heart rate occurred within the CS interval. By contrast, a large elevation in heart rate occurred within the UCS interval. On most trials, heart rate approached baseline levels sometime during the 5-min post-stimulus interval, although these post-stimulus responses became quite variable. With repeated daily exposure, the peak change in heart rate to the UCS became slightly smaller indicating possible adaptation to the noise. Fig. 2 summarizes these data. Heart rate adjustments in UCS alone trials were not significantly greater than those in CS–UCS trials and the trend toward smaller, acute responses was similar under both conditions.

![Graph](image-url)

Fig. 2. Exp. 1: 30-day trends in acute cardiovascular responses of Rhesus monkeys to signaled noise bursts. Conditioned stimulus was a red light. Unconditioned stimulus was noise burst at 112 dbA. Values of response change are shown on the ordinates and relevant measurement periods are shown on the abscissae. Changes are not statistically significant.
Blood Pressure, Acute Responses: Blood pressure also exhibited a slight decrease during the CS interval and then an increase associated with the UCS. This response, however, was uniformly delayed until about 10 sec after termination of the UCS. Toward the end of the experimental period, the magnitude of blood pressure adjustments became very small during presentation of either CS or UCS. Peak changes, that is, those occurring 10 sec after the stimulus was terminated, however, were not significantly reduced (see Fig. 2).

Baseline Trends: Perhaps the most important finding associated with this first experiment is that baseline blood pressure (sampled before either CS or UCS presentation) increased progressively over the 30-day interval (see Fig. 3). The overall averaged blood pressure increase was 28% (p < .001).

![Graph showing blood pressure changes over time](image)

Fig. 3. Exper. 1: Chronic trends in cardiovascular functioning: solid lines represent absolute value of peak, acute response to UCS, while dashed lines represent absolute value of "quiet time" response. Increases in systolic and diastolic blood pressure over a 30-day period are significant (p < .001).

Unlike blood pressure, baseline heart rate did not continue to increase during the same period, despite a precipitous change in the first half of the experiment. Baseline heart rate trends, however, hint at a form of cyclicity in which there is a rhythmic rise and fall of responses coupled with a failure to return to earlier
baseline levels (see Fig. 4).

![Graph showing heart rate response over days](image)

**Fig. 4.** Chronic trends in cardiovascular functioning over a 30-day period, no significant changes in heart rate occurred during the final 12 days of exposure.

**Discussion.**

Our baseline results are consistent with those of Forsythe (82) who found that, using long-term intermittent stress involving shock avoidance, blood pressure remained elevated even at times when the stress was not present. In general, previous work in other laboratories has established that the short-term elevation in blood pressure which occurs in response to repeated presentation of aversive stimuli can be associated with long-term elevations in baseline blood pressure. Such shifts in cardiovascular functioning are typically seen in experiments which use peripheral electric shock as aversive stimulus, (83—85) While shock is an excellent laboratory tool, it is hardly a widespread environmental pollutant. Our findings are of interest because changes similar to those which have been produced by shock have now been produced by noise, an aversive stimulus to which we are all exposed.

Our results also bear upon the significant environmental question of whether or not physiological adaptation eventually occurs to noises which initially produce startle reaction or discomfort. We found, for example, a generalized reduction in the magnitude of change in acute responses during UCS presentation as the experiment progressed. Averaged blood pressure responses taken over the final 12 days of the experimental period, in fact, showed very little or no elevation during the UCS interval. Such data might be interpreted as upholding the position that physiological adaptation does occur even to severe acoustic stimulation. Nevertheless, the perseveration of elevations in immediate post-stimulus blood pressure and the large increase in the absolute levels of baseline blood pressure seem to provide an effective counter-argument to this position.
It may also be speculated that these trends are not necessarily contradictory. Each may have been mediated by a different control mechanism. Adaptation during the relatively brief prestimulatory interval, for example, may reflect fast acting, neurogenic influences while post-stimulus perseveration and baseline sensitization may reflect slower acting, but longer lasting, hormonal influences. Whatever their basis, the important point is that the sustained adjustments did occur which apparently shifted the operational characteristics of the cardiovascular system upward. Whether ultimately adaptive or mal-adaptive, they represent an attempt by the animal to cope with a new and possibly threatening environmental factor.

EXPERIMENT II

Few individuals would be exposed to the level and character of the stimulus we used in the first experiment. We therefore initiated a second experiment designed to trace over an extended period cardiovascular responses to a noise recording which emulates more closely actual environmental conditions.

Methods and Procedures.

As in Exper. I, or subject was a young, female Rhesus monkey of about 4 kg weight. The proximal sensor was again a cannula placed in the abdominal aorta via the femoral artery. S was restrained and placed in the sound proof booth immediately after surgery. Surgical recovery took place within this context.

Baseline data was accumulated for 36 days until stable blood pressure and heart responses were achieved over a 9-day period. A protracted acclimatization period was necessary because an obviously painful breakdown and infection of this monkey's ileal catheater was associated with a steady rise in blood pressure which lasted for almost 4 weeks until we were able to instigate healing.

After healing progressed, blood pressure and heart rate returned to the desired stable levels. A high level of cleanliness coupled with the placement of sanitary napkin pads beneath the ileal catheater undoubtedly contributed to the rapidity with which a recovery was made in this monkey.

Once stable baseline responses were established, the monkey was exposed to noise for 12 hrs each day for 30 days.

We carefully specified the stimulus for this experiment. It consisted of a 20-min endless loop recording of daytime exterior noise made in the vicinity of Jackson Memorial Hospital, Miami, Florida. The sample was meant to represent, with regard to both source and level, typical inner-city, urban noise. Major noise sources were 154 ground vehicle passes (autos, trucks and buses) and 6 aircraft flyovers, as well as several nearby conversations, whistles, horns and tire squeals.

For experimental purposes, the level of the recording was adjusted upward about 5 dBA so that it exceeded 68 dBA 90% of the time, 76 dBA 50% of the time, and 84 dBA 10% of the time (see Fig. 1). The average equivalent noise level (Leq) was 78 dBA. The total noise environment of the animal was such that a large majority of the humans so exposed would express dissatisfaction.

The best evidence available, however, indicates that these levels will generally not lead to irreversible changes in auditory function in humans.
The above analysis was performed in the sound attenuating booth with the sound level meter placed at the level of the monkey's ear.

**Results.**

Fig. 6 shows the blood pressure responses for the last 39 days of Exper. II. As in the Exper. I, the data present arguments for and against cardiovascular system adaptation to noise. For example, subsequent to the commencement of daily stimulation, blood pressures became elevated well above control levels. During the first 10 days of the exposure period, nighttime (i.e., quiet time) blood pressure remained about as high as daytime blood pressure. Thereafter, however, the former declined and hovered close to baseline levels for the remainder of the experiment. Daytime levels by contrast remained elevated for the rest of the experiment.

Since measurements were taken on an hourly basis, we have been able to assess diurnal variation in cardiovascular function during the pre- and post-exposure periods. In the "natural" rhythm of heart rate and systolic blood pressure in this particular animal, both parameters reached maximum values around mid-
Fig. 6. Expos. II: 30-day trends in blood pressure responses to 12 hr per day community noise stimulation. Note initial increase in both daytime and nighttime blood pressure followed by a return of nighttime level to baseline and continued elevation of daytime levels.

morning and minimum values occurred during the early morning hours. The diurnal rhythm during the period of noise exposure was grossly similar to that for the control period, except that, hour for hour, the absolute values of systolic blood pressure were always higher.

By subtracting the hourly values associated with the pre-exposure period from the hourly values associated with the pre-exposure period, the effects of noise, independent of diurnal rhythm, can be determined.

As Fig. 7 indicates, the peak noise effects on systolic blood pressure began around 0500. It is important to note that this is 1 hr before the noise was turned on each day. The effects lasted until roughly 1400, and thereafter, fell to a minimum around midnight. A similar pattern was obtained for heart rate, although the major peak occurred at about 0900 to 1200 with a secondary one again at around 0500.

The far more rapid buildup of both systolic blood pressure and heart rate during the early morning hours, 0200 to 0500, in the pre-exposure period compared to the static or opposite trend during the pre-exposure period, indicates the possibility of an anticipatory response on the part of the animal. Such responses are said to enhance the perception of noise as noxious or annoying by humans.

Finally, as seen in Fig. 7, both heart rate and systolic blood pressure appear to be less affected as the noise continued throughout the day. Systolic blood pressure adjustments began to decline after 1400, and heart rate adjustments
declined after 0900. It is possible that this particular decrease in noise effect reflects the daily buildup of an increase in auditory threshold. The low effective intensity of the stimulus makes it seem unlikely that the adaptation of cardiovascular adjustments is based on a decline of sensory function, however.

EXPERIMENT III

Forrythe, (84) Augenstein (83) and others have demonstrated that chair restraint per se has little sustained effect on gross cardiovascular function in the Rhesus monkey. However, in order to demonstrate the innocuousness of our particular procedures, we chair-restrained an animal for 190 days. It was planned that the animal would be exposed to no unusual conditions, including no unusual noise.

In Fig. 8, the daily average heart rate for the period of restraint is shown: 80% of the daily averages fall between 150 and 170 beats/min. Interestingly, the two peaks in heart rate, which occurred about Days 110 and 150, were associated with heavy construction noise and vibration within our building. Other than these two changes, no sustained trend was apparent.

After 190 days, the catheter was removed and the monkey returned to its cage where it remains active and healthy as of this writing.

III. CONCLUSIONS

Our preliminary experiments demonstrated that both unexpected and continuous noise when presented repeatedly or chronically bring about sustained alterations in the balance of cardiovascular function which may become stimulus independent. The results are not definitive, but they do suggest to us that noise
may be one of the factors contributing to the long-term development of cardiovascular pathology in man. Of particular interest in this regard, are the sustained blood pressure elevations which may be induced by moderate but protracted noise stimulation.

Today, "cardiovascular diseases constitute the main underlying cause of mortality and morbidity in the United States." (86) One form of cardiovascular disease, hypertension, is the most common chronic disease. It is said to afflict between 10 and 15% of all Americans (87) and fully one-quarter of those above the age of 55. (88) Still, the pathogenesis remains unknown in 80–90% of these cases.

There are many lines of conflicting and contrasting evidence surrounding the mystery of "essential" hypertension, but perhaps the least contested fact is that the elevated blood pressure associated with hypertensive disease contributes in a striking way to disease and death. (89)

Despite universal agreement that hypertension is a widespread and serious — even life-threatening — problem, confusion remains as to whether "it" is a single disease entity (90) or various diseases subsumed under a single label. (91) It is not surprising then that there is also considerable disagreement regarding the underlying causes of hypertension. (91,92) Traditionally, a variety of systemic mechanisms (93, 94) has been proposed as etiologic factors, but more recently, the importance of certain situational, psychological and environmental effects have been amply documented. (95–98)

Perhaps the studies most pertinent to our present research have established that the etiology of hypertension can also be viewed from a larger, socio-environmental perspective. There is now "abundant evidence that social stress plays an important role in accelerating the progress of hypertensive illness." (99) Take as a case in point the often noted observation that essential hypertension is more prevalent and more severe in blacks than in whites. American black men under 50 have a death rate some 6 to 7 times greater than American white men of the same age. (100) The relation between skin color and blood pressure level, the
common patterns of blood pressure in certain isolated populations and in the
descendants of African tribes and the frequent occurrence of hypertension in
both maternal twins have been offered as substantiation for a genetic basis of
hypertension. (90) That such a position is probably not entirely accurate is
made clear by recent observations that:

a. poor blacks and poor whites of low socio-economic status, living in inner-
city areas, have higher blood pressure on the average than do their richer, high
status, suburban counterparts;

b. blacks who were born in the inner city have higher blood pressure on the
average than do blacks who migrated into these areas; and

c. black adolescents attending suburban schools have lower pressure than do
those who attend inner-city schools. (101—103)

Such findings suggest at the very least an environmental overlay on the patho-
genesis of essential hypertension.

There are many ways, both obvious and subtle, in which inner-city areas dif-
fer from suburban and rural areas. The distinguishing characteristic which is
most germane to the issues raised by our study, however, relates to the fact that
excessive noise seems to be an ever-present by-product of urban activity. It has
been shown that downtown areas are about 10 to 15 dBA noisier than quiet sub-
urban areas. (104)

High blood pressure appears to co-exist with higher noise in the inner city
and with noisier occupations. This fact, together with evidence from many
studies, including our own, strongly suggest that noise has profound effects on
cardiovascular function, particularly in aspects relating to blood pressure regula-
tion. Unfortunately, present lines of evidence do not specify the nature of
these effects conclusively. Therefore, we believe further inquiry is vitally impor-
tant.

The procedures developed in our preliminary studies should enable us to ex-
amine cardiovascular adjustments to long periods of noise exposure in both
semi-restrained and unrestrained monkeys. We believe the course and site of
these adjustments, if they occur, can now be described in detail in a species
closely related to man.

IV. SUMMARY

Considerable evidence has accumulated which bears on the non-auditory as-
pects of noise effects. Because of the serious and pervasive nature of cardio-
vascular disease, including hypertension, the relation between noise and cardio-
vacular function has received particular attention. For a variety of reasons, how-
ever, past efforts in this area have not provided a satisfactory understanding of
man's long-term reaction to noise.

In the preliminary studies reported here, an attempt was made to overcome
some of the deficiencies inherent in previous work: first, by studying cardio-
vacular responses in a species closely related to man, and second, by maintaining
rigorous and continuous control of the acoustic environment of the subject.

We exposed one semi-restrained Rhesus monkey to unpredictable, intense
noise bursts and another to recordings of exterior community noise. Cardio-
vascular adjustments, measured by an indwelling catheter, persisted or became
sensitized to certain aspects of the experimental situation and adapted to other
aspects. In Exp. I, acute blood pressure adjustments to the noise bursts became
slightly smaller as the stimuli were repeated over a 30-day period. Baseline blood
pressure measured during quiet times rose by about 30% over the same period;
however, in Exp. II, prestimulation blood pressure remained elevated for more
than 30 days to recorded community noise at an Leq of 78 dBA, while non-
stimulation blood pressure fell to baseline after 10 days. Exp. III explored the
long-term effects of our restraining techniques alone; no discernible trends in
cardiovascular responses were noted over a 190-day period.
These results offer suggestive evidence that noise may have a profound effect
on cardiovascular function, particularly blood pressure regulation.

ACKNOWLEDGEMENT
The valuable technical assistance of E. Greenfield, A. Winer, S. Goldberg,
A. Kelly, M. Szpak, E. Shapiro, and M. Vellon is gratefully acknowledged.
Special thanks are due Mr. R. Sieffert.

GENERAL BIBLIOGRAPHY
2. Chalupski, J.D. (Ed.), Transportation Noise, A symposium on acceptability criteria.
3. Cohan, A., Airport noise: some homes and public health. Proc., 1st Conf. on Aircraft
5. Chapman, R.J., Mcclain, S.C., and Harris, R., Relation of noise measurements to
7. Lipez|nsha, D.M., Theoretical considerations in the use of high frequency hearing loss in
children. Progr. Int'l. Congr. on Noise as a Public Health Problem, Dubrovnik, Yugo-
slavia, 1973. (American ref?)
1968, 90, 543-555.
389-393.
USPSA Rep., No. 150/7/73-092.
a Public Health Hazard, Ward, W.D., and Pricie, J. (Eds.) ASHA Reports No. 4, 37-39,
1969.
25. Aminoff, A. Azura D-RNA changes in the adrenal and cerebral cortex of rats exposed to
26. Gentilesco, Y.P. Noise induced changes in the adrenals. Gipsana I Sanita, 1959, 34,
147-161.
male albino rats subjected to experimental stress. Biochem. Pharmacol., 1966, 15,
1707-1713.
28. Anthony, A. and Ackerman, E. Effects of noise on the blood eosinophil level and ad-
29. Ogla, C.W. and Lottstott, M. The urinary changes induced by high sounds (20 keyw.)
30. Rothchild, H.D. and Shew, S.M. Urinary sodium/potassium ratio determined by tracer
31. Friedman, M., Byers, B.G. and Breen, A.E. Plasma lipid response of rats and rabbits to
32. Done, W.F., Balsehendran, N.K. and Maclun, G. The level of vanillylmandelic acids
and 17-ketosteroids in women working in a noisy environment. J. Acoust. Soc. Am., 1974,
56, 1297-1300.
33. Jackson, H. and Kronerovka, B. Studies of the influence of noise on the general health of
workers in large engineering factories, an attempt at evaluation. Cited in: Public Health
34. Lippinsbort, D.M. Indicators of environmental noise. In: Indicators of Environmental
35. Ando, Y. and Hattori, J. Statistical studies on the effects of intense noise during human
36. Singh, K.B. and Rao, P.B. Studies on the polyvastic ovaries of rats under continuous
37. Kryst, K.D., Jansen, G., Parker, S., Paver, E., Risen, G. and Williams, H.L. Non-
38. Khazinovich, M.L. The effect of noise on the general immunological reactivity of the
32, 2101-2104.
40. Molema, M., Lassere, F. and Smith, L.W. Effect of auditory stress upon methylene-
41. Cizek, O., Franquegull, A. and de Campora, E. Experience with acoustic sensory stress V.
behavior of sodium and potassium in the rat submandibular gland in auditory stress,
42. Ivanov, Z. Changes in blood oxygenation rate due to intense noise and to vibration.
Trudove Na Uzucmo-Uzucovalejska Institut Po Ohranu Na Truda i Professional'nye
Zabolokatya, 1969, 1, 1, 67-70.


47. Savvov, G.A. Cortical and mesencephalic reticular formation function after prolonged exposure to pulsed and steady-state noise. Zh. Vyschet Nervnui Drevatsloboi, 1972, 6, 103-110.


66. Shevlev, M.N. and Murov, M.A. The influence of intense noise and neurophysiological tension on the level of arterial pressure and the incidence of hypertensive vascular disease.


78. Sauti, W. and Madake, J. Phasic changes in heart rate following acoustic stimuli during natural human sleep. Pfugers Arch., 1964, 294, 165-175.


89. Lewis, E.A. High blood pressure, other risk factors and longevity: the insurance viewpoint. In: Ref. 88.

100. Freis, E.D. Age, race, sex and other indices of risk in hypertension. Pp. 31-41 in (see Ref. 83).
## SUMMARY FORM FOR

**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tbody>
<tr>
<td>E.A. Peterson (Chief, Div. of Auditory Research) et al.</td>
<td>University of Miami School of Medicine, Dept. of Otolaryngology, Division of Auditory Research, Miami, Florida</td>
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<th>English</th>
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<table>
<thead>
<tr>
<th>Type &amp; duration of experiment</th>
<th>Purpose for study</th>
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<tbody>
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<td>controlled laboratory</td>
<td>to measure the effects and after-effects of noise on heart rate of an unrestrained animal</td>
<td></td>
</tr>
<tr>
<td>4 mo.—control period—quiet</td>
<td></td>
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<tr>
<td>7 weeks—test period—noise</td>
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<tr>
<td>4.5 mo.—control— maxlen</td>
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<tr>
<th>Description of test groups (subjects, age, sex, etc.)</th>
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<tbody>
<tr>
<td>1 female Rhesus monkey, 4.5 kg, with no prior exposure to loud noise, unrestrained</td>
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<th>Control of other stressors</th>
<th>Statistical Methods</th>
<th>Nonauditory effects</th>
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<td>laboratory conditions</td>
<td>average daily standard error of the mean</td>
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<td>CVS Response Measured</td>
<td>heart rate—EKG</td>
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<tr>
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<tr>
<td>spectral characteristics: described in previous paper</td>
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<tr>
<td>noise level: Leq=79.4 dBA</td>
<td></td>
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<tr>
<td>length of exposure: 12 hour per day</td>
<td></td>
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<tr>
<td>length of test: 6 m—6 hom</td>
<td></td>
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<tr>
<td># of trials: 1 daily for 7 weeks</td>
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| Author's conclusions | Prolonged exposure to moderate noise levels affects heart responses both during stimulation and for a period of several months afterward. Pathological cardiovascular change could thus begin prior to significant acoustic trauma. |

<table>
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<th>Evaluation &amp; comments</th>
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<tr>
<td>(1) It is curious that blood pressure, which showed the greatest response to noise in the first experiments (published in the J. Auditory Research 15:234—251, 1975) was not monitored in this follow-up study.</td>
<td></td>
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<tr>
<td>(2) the monkey had a cardiac conduction defect resulting in missed beats, which may have affected the results</td>
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The effect of a moderate noise stimulus on heart rate was studied in a 4.5 kg. female Rhesus monkey. The animal had electrodes and a radio transmitter for heart rate monitoring implanted with a vest and a backpack assembly to allow free movement (restraint alone was found to cause significant overall changes in heart rate in a previous study). A four month quiet control period in the acoustic test chamber was included to establish baseline heart rate levels in the monkey. The monkey was then exposed to a noise stimulus of recorded urban noise with an average energy level, L eq, of 79.4 dBA (over 68 dBA 90% of the time) for 12 hours per day for 7 weeks. This noise level is below that which could cause acoustic injury in the Rhesus monkey. The noise exposure began at 6 a.m. each morning. Daily heart rate rhythms (diurnal rhythms) were altered in the early morning hours by the noise. Heart rate increased significantly during this time period. An anticipatory response to noise was indicated by the presence of near peak effects on heart rate one hour before the noise began. Neither heart rate nor the early morning rhythms returned to normal baseline levels in a 4.5 month post-stimulus period. Moderate noise levels can cause significant cardiovascular changes prior to any acoustic changes.
CONTINUING STUDIES OF NOISE AND CARDIOVASCULAR FUNCTION

E.A. PETERSON, J.S. AUGENSTEIN and D.C. TANIS
Division of Auditory Research, Department of Otolaryngology
University of Miami School of Medicine, Florida

A substantial amount of evidence derived from both animal and human experimentation indicates that prolonged exposure to high levels of noise may be associated with various forms of cardiovascular dysfunction (Anticaglia and Cohen, 1970; Welch, 1973; Miller, 1974; Hattis et al., 1976). Nevertheless, a clear and unequivocal relationship between noise per se and cardiovascular function has yet to be established. Certain deficiencies which characterize work in this important area contribute to the unsatisfactory state of our knowledge. Among them are selection of inappropriate animal models, relatively short-term measurement periods and inadequate specification and control of noise exposure (Smookler, Goebel and Siegel, 1973; Lockett and Marwood, 1973; Rosecrans, Watzman and Buckley, 1966; Yeskel et al., 1948; Cartwright and Thompson, 1974; diCantogno et al., 1976; Smookler and Buckley, 1969; Jonsson and Hansson, 1977; Parvizpoor, 1976; Andriukin, 1971; Kavoussi, 1973).

Recently, my colleagues and I performed a series of experiments which overcame some of the problems encountered in earlier work. For example, we chose as an animal model a species closely related to humans and we carefully specified the noise conditions to which each subject was exposed (Peterson et al., 1975). We demonstrated, with chair-restrained rhesus monkeys, that changes in heart rate and blood pressure can occur in association with protracted exposure to both short bursts of intense noise, presented randomly throughout working hours, and to moderate levels of community noise, presented for 12 hours daily.
Despite provocative findings, these pilot studies were themselves open to criticism. For example, although it improved stimulus control, partial restraint imposed unnatural limits on the animal's freedom to move in space and, therefore, may have caused additional, uncontrolled stress. Further, no attempt was made to follow the course of cardiovascular function after the period of exposure had been completed. We did not, in other words, study the long-term after-effects of noise exposure.

We are presently conducting an experiment which deals with these specific criticisms; that is, we are monitoring an index of cardiovascular function (heart rate) in an unrestrained animal and are continuing the measurements beyond the period of noise exposure. We hope, thereby, to provide a clearer, though still incomplete, understanding of the relationship between noise and cardiovascular function. This report is a summary of our progress to date.

METHOD

Subject

The subject for the experiment is a young, 4.5 kg female rhesus monkey named Jean. Although used in an earlier experiment as a control animal, she had not been exposed to any unusual noise conditions prior to serving in this study.

Stimulus conditions

The stimulus has been described in detail elsewhere (Peterson, Augenstein and Hosek, 1976). Briefly, noise was recorded for 30 minutes on a sidewalk adjacent to Jackson Memorial Hospital, a major community hospital in Miami, Florida. Included in the sample are about 150 bus, truck and automobile pass-bys, a half-dozen aircraft fly-overs, numerous conversations, shouts, horns, whistles and brake squeals, as well as incidental noise. The sample was designed to be illustrative of inner city, daytime community noise.

Actual recorded levels are increased by about 5 dB to a level just below
that which might eventually cause acoustic trauma in the rhesus monkey (Luz et al., 1973). As can be seen in Figure 1, levels exceed 84 dB(A) 10% of the time, 75 dB(A) 50% of the time and 68 dB(A) 90% of the time. Average energy level, $L_{eq}$, is 79.4 dB(A). These values are accurate only if there is a constant relationship between subject and source.

*INSERT FIGURE 1 HERE*

Because in this experiment the monkey is allowed full freedom of movement within the 1m x 1m x 2.5m test chamber, its actual exposure to the noise can only be approximated. The problem is further complicated by the use of highly reflective surfaces which are necessary to maintain hygienic conditions within the chamber. Thus $L_{eq}$ measured at 8 different points within the chamber was found to vary by about ±4.0 dB(A).

During the noise exposure portion of the experiment, the animal was subjected continuously to the community noise sample for 12 hours, from 0600 to 1800, each day. Steady operation of a ventilating fan provided a "noise floor" of about 52 dB(A). Its operation also provided masking for extraneous sounds.

**Procedure**

The animal is lightly anesthetized with Sernylan\textsuperscript{R} (0.11 ml/kg I.M.). Prophylactic Cephadyl\textsuperscript{R} is also administered (0.5 gms I.M.). Two strands of 00 stainless steel suture material, sewn into the skin above the ascending aorta and the cardiac apex, serve as electrodes. The electrode leads are connected to a radio transmitter\textsuperscript{1} mounted within the rear pocket of a protective vest. The vest and backpack assembly is light enough to allow almost complete freedom of movement and when fitted properly, causes no apparent discomfort to the monkey. Upon recovery from anesthesia, the

\textsuperscript{R} Sernylan is a registered trademark of Parke, Davis and Company.

\textsuperscript{R} Cephadyl is a registered trademark of Bristol Laboratories.

\textsuperscript{1} "Saturn 3" Space Labs, Inc., Los Angeles, Cal.
animal is placed in the test chamber which is furnished with various perches and swings to encourage a normal amount of gross bodily movement.

Analog EKG information is telemetered to a processing receiver that is connected to a second monitor which provides a hard copy print-out of both EKG waveforms and continuous heart rate trends for off-line analysis.

Transmitter batteries last about 30 days. When it becomes necessary to replace them, the animal is again anesthetized, the vest is removed and washed, the electrodes are inspected and the monkey is washed and examined for skin lesions.

RESULTS

Trends in daily heart rate

Our findings are summarized in Table I and Figures 2 through 5. Baseline heart rate levels in this animal were established daily for 4 months prior to the noise exposure period. Since, as noted above, she had previously served as a chair-restrained control subject, it became possible to compare, in the same animal, long-term heart rate trends under conditions of quiet and restraint with those under conditions of quiet and non-restraint.

When chair-restrained, average daily heart rate for this animal was 150.1 Beats Per Minute [BPM]. An index of variability, the average of daily standard errors of the mean, was 1.24 BPM. As shown in Table I, when free-moving, average daily heart rate was 140.8 ± 2.85 BPM. The difference is significant (p < .01). Contrary to earlier reports (Forsythe, 1969; Augenstien, 1974), then, chair-restraint does indeed seem to affect overall heart rate levels. In keeping with the findings of the above reports, however, no discernible trend is evident either during the 6-month restraint or the 4-month non-restraint period. This is clearly shown for

1 "Saturn 3, Space Labs, Inc., Los Angeles, Cal.
2 "Cardio-Care EBD, American Optical Co., Bedford, Mass."
the latter period in Figure 2.

Following the establishment of a baseline, the animal was exposed 12 hours per day to the noise sample. Over the next 24 days, daily heart rate increased significantly \((p<.01)\) to an average 157.0 ±2.10 BPM.

At this point, the radio transmitter malfunctioned. While it was being repaired, the subject was maintained on the same 12-hour per day exposure schedule. Within a week, the backpack had been re-installed and the entire system re-calibrated. Measurements then resumed on a continuous 24-hour basis. During this hiatus, however, heart rate had dropped significantly \((p<.01)\) and for the following 21 days it remained at an average 113.3 ±3.00 BPM.

At the end of the exposure period, the vest and transmitter were removed and the animal allowed to move about in the chamber unencumbered for approximately 6 weeks.

At the end of that period, heart rate measurements under quiet, non-restraint conditions were resumed and continued for the next 3 months. During this time, only a gradual recovery in heart rate has been observed. In fact, at 4.5 months, post-exposure heart rate still has not returned to baseline levels.¹

This particular monkey suffers from a cardiac conduction defect which is manifested as an occasional generation of P-waves without subsequent generation of QRS complexes. Such a pattern appears to be consistent with a second degree atrio-ventricular block in which "... all QRS complexes are preceded by P-waves, but not all P-waves are followed by QRS complexes" (Berne and Levy, 1972). We have labeled this conduction defect simply as "missed beats." The percentage of missed beats

¹ Note on the right of Figure 2 that during the course of recovery, the animal developed a fibrous growth on her arm which appeared to cause her considerable discomfort. While the lesion was growing and immediately subsequent to its removal, heart rate increased significantly.
throughout the experiment is indicated in Table I. Note that the percentage is inversely related to overall heart rate. Fewer missed beats are generated with increased heart rate. Note also that for the pre-exposure baseline period, missed beats averaged about 11% of total beats during the daytime hours. Within the first half of the noise exposure period, when heart rate was significantly higher than baseline levels, missed beats accounted for only about 2% of the total. During the second half of the noise exposure period, when heart rate was significantly lower than baseline levels, missed beats increased significantly to 24%. As recovery proceeded, missed beats decreased to 16% during January and February and to about 13% throughout the month of March. This is close to pre-exposure baseline levels.

Trends in diurnal rhythm

It should be evident from the heart rate and missed beat trends described above that in this experiment, noise exposure after-effects are not short-lived. Perhaps the most striking demonstration of this fact, however, is derived from changes in the diurnal rhythm of heart rate. An illustration of how noise affects this aspect of rhythm is shown in Figure 3. These data are derived from an experiment in which a different chair-restrained monkey, but the same community noise sample, was used. In the top half of the figure, diurnal rhythm for heart rate before and during noise exposure is indicated.

INSERT FIGURE 3 HERE

Throughout the pre-exposure period, heart rate remained relatively constant during the early morning hours, 0100 to 0600. During the exposure period, it increased steeply.

By subtracting hourly heart rate values associated with the exposure period from those associated with the pre-exposure period, it became possible to determine the effect of noise independent of diurnal rhythm. In the bottom half of Figure 3, it can be seen that near-peak noise effects were reached at 0500, one hour
before onset of daily noise. This rapid build up resembled what would, in humans, be called anticipation. Although in the present experiment we did not initially measure heart rate each hour, confirmation of this "anticipatory" response eventually became possible.

Clearly, heart rate varied systematically throughout the day. As shown in Figure 4, heart rate for this animal is generally lower during the late evening and early morning hours than it is during midday under all the conditions we have studied, including pre-exposure restraint, per-exposure non-restraint and post-exposure non-restraint. There are, nevertheless, significant differences among the various conditions. These are illustrated in Figure 5.

Return to a diurnal pattern similar to that which characterized the pre-exposure era for this animal would seem to be a reasonable indication that noise exposure after-effects have subsided. While this animal was in quiet and restrained (in 1975), the daily heart rate cycle reached a low point between 0500 and 0600. Slope of a line fitted to data points is steeply negative for the hours 2400 to 0600. In contrast, while this animal was exposed to noise and was unrestrained, quite a different trend was manifested; that is, a heart rate minimum was reached near 2400 hours and slope of the line fitted to data points is steeply positive for the hours 2400 to 0600. This trend reinforces the notion that some sort of anticipatory response is associated with daily onset of the noise. Six to ten weeks after the noise exposure period (labeled "JAN" in Figure 5), slope of the fitted line is still positive. After 10-14 weeks (FEB.), slope of the fitted line is about zero, and after 14-18 weeks (MAR.), it has become slightly negative.
DISCUSSION

The noise-induced changes in early morning diurnal rhythm observed in the present experiment are consistent with those changes observed in a previous experiment in which the animal was restrained.

The course of changes manifested in overall heart rate, proportion of missed beats and diurnal rhythm clearly indicates that prolonged exposure to moderate noise levels not only influences cardiac function during the period of stimulation but also that these influences dissipate slowly after the exposure has ended. The protracted recovery process shown thus far for this monkey may in fact never be completed.

A failure of cardiac function to return to baseline levels may provide the opportunity for resolving a paradox implied in recent studies. It has been a common assumption that if noise cannot be heard, it cannot have significant physiological effects. Investigators consistently report, however, that industrial workers with greater hearing loss appear to have a higher incidence of cardiovascular dysfunction. Based on our present findings, the possibility arises that pathological cardiovascular adjustments are begun before audiologically significant acoustic trauma occurs. These adjustments may in fact contribute to hearing loss usually ascribed solely to noise.

Some comments should be made regarding the puzzling downward shift in heart rate which took place during the final three weeks of the noise exposure period. Physical discomfort on the part of the monkey, malfunction of the monitor and changes in feeding or cleaning schedules have all been eliminated as possible sources for this drastic alteration in cardiac dynamics. It is difficult to specify the physiological factors responsible for the abrupt change, however, in the absence of other measures of cardiovascular response. Since under present circumstances these
are impossible to obtain, it can only be speculated that perhaps during the first weeks of noise exposure, sympathetic-adrenergic influences governing the greater heart rate were severely diminished, thereby inducing parasympathetic-cholinergic dominance. This was manifested, in turn, by lowered heart rate.

A second possibility is that heart rate and blood pressure were elevated simultaneously. At some point, in order to maintain cardiovascular homeostasis, it became necessary to compensate for the increase in blood pressure.

**SUMMARY**

Long-term trends in heart rate were observed in a free-moving rhesus monkey.

Initially, normal heart rate for the animal was defined over a span of 4 months. She was then exposed to a community noise recording 12 hours per day for 7 weeks. Pre- and post-exposure changes were noted in several aspects of cardiac function.

During the exposure period, heart rate and proportion of aberrant EKG responses rose significantly above, and then, fell significantly below baseline levels. Patterns of diurnal rhythm for early morning heart rate were also altered by noise exposure. These last results are consistent with those of an earlier study in which a restrained animal had been subjected to similar stimulus conditions.

After-effects of the single, prolonged noise exposure episode have dissipated gradually. During the course of a 4-month post-exposure period, heart rate and proportion of aberrant EKG responses have slowly returned to near-baseline levels.

Early morning diurnal rhythm for heart rate has not yet returned to its pre-exposure pattern.
REFERENCES

Andriukin, A. The level of arterial pressure and the frequency of hypertension in workers of noisy plants. Gig. Tr. Prof. Zabol., 1971, 5, 11-17.


ACKNOWLEDGEMENTS

The technical assistance of Manuel Solano, Heldo Gomez, Regino Gonzales, Catherine Popkin, Ben Teitelbaum and Jason Finder is gratefully acknowledged. Special thanks are due Richard Seifert and Sydney Iglitzen.
FIGURE CAPTIONS

FIGURE 1: Community Noise Experiment II: Statistical characteristics (L values) of stimulus.

FIGURE 2: Community Noise Experiment II: Daily average heart rate for unrestrained monkey, Jean. Heart rate trends after 11-3-76 were measured 24 hours per day. Note the precipitous increase then decrease in heart rate during 7-week exposure period.

FIGURE 3: Community Noise Experiment I: Restrainted monkey, "Mars". Top: Diurnal rhythm for heart rate during pre- and per-exposure periods. Bottom: Diurnal noise effects on heart rate. Near-maximum effects were exhibited 1 hour prior to onset of noise each day (adapted from Peterson et al., 1975).

FIGURE 4: Community Noise Experiment II: Trends in diurnal rhythm of heart rate during early morning hours. Recovery from 6-week exposure to community noise sample may be manifested by gradual return to negative slope of curve for these hours.

FIGURE 5: Community Noise Experiment II: Diurnal rhythm of heart rate. Absolute values have been converted to percentage of maximum daily heart rate for normalization. Note truncated dynamic range of heart rate under restraint, no-noise conditions and increased dynamic range of non-restraint, noise exposure conditions (thick, solid line). Generally, the course of diurnal rhythm follows a bell-shaped curve. Peaks during the day are associated with feeding and other activity near the test chamber.
TABLE I
Heart Rate Trends in Unrestrained Monkey, Joan

PRE-EXPOSURE
(6/9/76 - 10/1/76)
\[ \bar{x} = 140.8 \]
\[ s_{\bar{x}} = 2.85 \]
Missed Beats** = 11%

PER-EXPOSURE

|  |  
|---|---|
| I (10/2/76 - 10/26/76) | II (11/3/76 - 11/23/76) |
| \[ \bar{x} = 157.0 \] | \[ \bar{x} = 113.3 \] |
| \[ s_{\bar{x}} = 2.10 \] | \[ s_{\bar{x}} = 3.00 \] |
| Missed Beats = 2% | Missed Beats = 24% |

POST-EXPOSURE

|  |  
|---|---|
| I (1/6 - 2/1/77) | II (2/3 - 2/10) |
| \[ \bar{x} = 129.8 \] | \[ \bar{x} = 134.6 \] |
| \[ s_{\bar{x}} = 3.54 \] | \[ s_{\bar{x}} = 2.91 \] |
| Missed Beats = 15% | Missed Beats = ? |

III (2/10 - 2/21) Fibroma growth and removal
\[ \bar{x} = 150.8 \]
\[ s_{\bar{x}} = 2.54 \]
Missed Beats = 7%

IV (2/22 - 3/13)
\[ \bar{x} = 133.6 \]
\[ s_{\bar{x}} = 2.89 \]
Missed Beats = 13%

V. (3/29 - 4/4)
\[ \bar{x} = 131.0 \]
\[ s_{\bar{x}} = 3.11 \]
Missed Beats = ?

** EKG waveform which may indicate a second degree A-V block.
* Measured during period 0900-1700 hours.
* Average daily standard error of the mean.
UNRESTRAINED MONKEY: JEAN
PRE-EXPOSURE (6/9-10/1/76)

EXPOSURE (0600-1800)
CORRECTED FOR 24 HRS.

POST-EXPOSURE (1/6-4/4/77)
FIBROMA GROWTH & REMOVAL

HEART RATE (BPM)

3 DAY—BLOCKS

02 04 06 08 10 12 14 16 18

02 04 06 08 10 12 14

02 04 06 08 10 12 14 16

110/2-10/26/76
II 11/3-11/23/76

24 HR. MEAN
**SUMMARY FORM FOR STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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</thead>
<tbody>
<tr>
<td>G.S. Zvereva</td>
<td>Scientific research Institute of Labor Hygiene and Professional Illnesses Donetsk, USSR</td>
</tr>
<tr>
<td>N.V. Reiner</td>
<td></td>
</tr>
<tr>
<td>A.V. Koliganov</td>
<td></td>
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**Investigator's Phone No.**

**Sponsoring Organization:**

Same as above

**Citation:**


English translation.

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<th>No. of Fig.'s 2</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>English translation.</td>
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</tbody>
</table>

**Type & duration of experiment:**

Survey of workers at various types of factories having rolling mill noise

**Purpose for study:**

To study the effects of rolling mill type noises (intermittent, percussive, impact noises) on workers

**Description of test groups (subjects, N, SEX, AGE):**

No test and control groups—a general survey of groups of workers was done in various factories; all workers were 30-40 years old and had 5-15 years of service (in this type of factory, presumably)

**Control of other stresses:**

No control

**Statistical Methods:**

Used to compare significance of differences in noise effects between groups of workers

**CVS Response Measured:**

Pulse

Blood pressure

**Nonauditory effects:**

CVS blood pressure was altered—elevated in many cases—exact changes not reported

Pulse—fluctuations noted due to noise

**Other: memory and attention:**

Physiological complaints were listed

**Author's conclusions:**

Physiological changes, such as increased or fluctuating pulse rate and blood pressure, decline in pain sensitivity, dizziness, as well as a decline in memory and attention, were observed.

**Evaluation & comments:**

The cardiovascular changes were not reported quantitatively, so no real conclusions can be drawn. The one interesting finding was that nonauditory physiological effects were observed prior to the development of auditory impairment.

Workers in various types of factories in the U.S.S.R. that have intermittent rolling mill noise were surveyed for auditory and non-auditory physiological effects, as well as psychological effects. Various trends in types of physical ailments in this industry (metallurgical plants) were reported, although very little quantitative data is included. Some very high intermittent noise levels were recorded, some as high as 122 dB. The 340 workers surveyed were 30 to 40 years old and had been working in the factories for up to 15 years. Hearing impairments were found in 168 (50%) of the workers surveyed. Physiological changes included neurological problems such as dizziness, numbness, and headaches and cardiovascular effects such as altered and often increased pulse rate and blood pressure. The psychological effects included a decrease in memory and attention. Many of the changes were observed even in the first year of work, before the development of any hearing impairment.
ROLLING MILL NOISE AND ITS EFFECT ON WORKERS

By G.S. Zvereva, M.V. Ratner and A.V. Kolganov

Gig. Sanit; (11), 1975

Scientific-research Institute of Labor Hygiene and Professional Illnesses, Donetsk.

According to the observations of a series of authors, the influence of intermittent noise on the human organism is different from the effect of stable noise. The intensity, the spectral make-up and the time parameters of the former in areas where cutting is going on, where they are trimming, stacking and where they are transporting different types of rolled stock are determined with the aid of a pre-calibrated loop oscillograph N-117 and an NIV-1 noise and vibration gauge. Before the start of a shift of 340 workers, subjected to the effect of intermittent noise, we measured the audibility threshold, the arterial pressure and the pulse, we determined the condition of pain, vibration and temperature sensitivity, and we actively identified their complaints. We also studied the function of memory and attention.

In pipe and sheet rolling plants in the areas around the pilger mills and cross-cutting aggregates, the noise is typically percussive, with almost instantaneous increase of the front part of the impulse (40 ms), with short interval vibrations (0.2 sec) and with high maximum levels (122 decibels). According to the intensity and the character of the intermittent noise generated by the stamping presses and the presses of the Pela type in a rail structuring mill and in rail-splicing plants, it is close
to that which has been described, but it differs in the lower frequency of the impulses (20 imp/min), it is somewhat greater, close to 100 msec, in the increase time of the front as well as the long length of the vibrations and pauses (within 1-2 sec). In section rolling plants, the noise emerging in the work of cutting cold and hot metal, may be referred to the noise of friction. It is characterized by a relatively long increase time of the front (more than 100 msec), comparatively long vibrations and pauses (several seconds), and consequently a low frequency (2 imp/min). In the case of the friction of rolled profiles around the cooling rack of the coolers while they are being transported with rolling skids, the noise of friction also emerges, but with a higher medium intensity of impulse (115 decibels). At the Communist metallurgical plant, the construction of rolling skids has been changed so that the metal does not touch the cooling rack of the coolers when it is moving; this did away with the friction of slip, thanks to which the intensity of the noise dropped 18 decibels in impulse and 20 decibels in average magnitude.

The noise generated in thick sheet rolling mills with disk and guillotine cutters, and also with the movement of metal sheets along roller conveyors is a combination of percussive noise and the noise of friction and is characterized by non-periodic, inconstant parameters. In some samplings, separate realizations are very essential to distinguish one from the other (100-150 times). Momentary values fluctuate from extremely unsatisfactory (intensiveness in the impulse up to
142 decibels, increase time 20 msec, pause period 0.1 sec) to relatively satisfactory.

Although an opinion exists concerning the great aggressiveness of non-periodic noises (G.A. Suvorov and L.A. Marakushkin), we did not observe this. Evidently, the reason for this is that in production, as opposed to office conditions in the case of the experiment, the alternating of impulses of a different intensity and a pause is caused by the course of the technological process and the appearance of an alternate, high-intensity stimulus (impulse) was not unexpected for the workers. At the same time, the amount of high intensity impulses is known to be less than for example in the case of percussive noise of comparable force. Evidently, this determined the lower activity of the non-periodic noise. Thus, among the 37 workers under conditions of non-periodic noise where the guillotine knives and disk knifes are located we found a hearing loss of an initial and slight degree, 43% according to the classification of V.E. Ostapkovich and N.I. Ponomarevoi. In the case of 25 workers at the pilger mills and cross cutting aggregates under the influence of percussive noise, the hearing changes were more profound: we found not only initial and slight, but also moderate impaired hearing and impaired hearing of a considerable degree (20%). The general number of those with impaired hearing in this group (84%) was also substantially greater than in the preceding group (P < 0.01). The stable noise, with an intensity of 105 decibels, equivalent to the average force of non-periodic and impact noise, brought about a
corresponding change in 68% of those examined (25 people). But
the age and the work stage of all the comparison groups were
similar: for the workers chosen to be examined 30-40 years old
and a service life of 5-15 years.

Influence of the production stage and the hearing condition
on the function of memory and attention (in % of the number
surveyed) (Table 1)

<table>
<thead>
<tr>
<th>Condition of the function of memory and attention</th>
<th>Service time (years)</th>
<th>Coefficient of ranked correlation with a hearing loss at stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memory decline:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>hearing:</td>
<td>Up 1-2</td>
<td>41.7 61.1 40.7 50.0 65.0 0 0.9±0.01</td>
</tr>
<tr>
<td>visual</td>
<td>8.4 11.1 14.8 10.7 42.7 0 0.9±0.01</td>
<td></td>
</tr>
<tr>
<td>Drop in attention concentration</td>
<td>16.7 22.2 33.3 32.1 60.9 0.6±0.03 0.9±0.01</td>
<td></td>
</tr>
</tbody>
</table>

Table 2.--Influence of the character of labor on the function of memory and attention (in % of the number surveyed)

<table>
<thead>
<tr>
<th>Condition of the function of memory and attention</th>
<th>Predominant component of labor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memory decline:</td>
<td>Physical</td>
</tr>
<tr>
<td>hearing:</td>
<td>50.0</td>
</tr>
<tr>
<td>visual</td>
<td>20.0</td>
</tr>
<tr>
<td>Drop in attention concentration</td>
<td>53.0</td>
</tr>
</tbody>
</table>

Note: the + sign on the figure is the presence of a direct, intimate correlation dependence on hearing loss (coefficient of the correlation r > 0.6).
188 men were found to have hearing losses (50%) at the described places of rolling mills. The initial degree of the decline among them was 23%, to a slighter degree 19%, moderate and great 9%. In addition to the impaired hearing, many workers were found to have changes in the neurological state. A rather large number complained of headaches, dizziness, irritation, not sleeping well, increased fatigue, pain in the region of the heart, heart palpitations, extended pain in the muscle extremities, cramps, a feeling of numbness, etc. The complex of such complaints was shown to be 25% of those studied after 3-5 years of work under noisy conditions. In many cases, the complaints were combined with objective symptoms. We noted a disruption of arterial pressure (often an increase of it), lability of the pulse, a decline of vibrational and pain sensitivity, up to complete anesthenia, thermoasymmetry, tremors of the hand and eyelids, instability of the Romberg posture.

Many of the listed changes, 17-45% of the cases were recorded on those surveyed already in the first year of work. In the same period, with 20% of the workers we noted a drop of more than 30 decibels in the perception of high tones with normal audibility in the speech range frequency. The early neurological symptom is the result of functional disorders of the central nervous system. Thus for the purpose of a well-timed exposure and taking dispensary accounting of such people, we required a doctor's examination and audiometric examination of the workers under noise conditions during the first year of work.
There was a certain interest in studying the workers' memory and attention functions. An analysis of different stages of work under noise conditions (Table 1) showed that visual memory and attention concentration got worse with an increase of the production stage and age and that there is a direct and close correlative connection with hearing disruption. We did not find a dependence of the condition of hearing memory on the character of the labor (Table 2). At the same time, the presence in groups of workers with a sufficient stage of close, direct correlation between a drop in hearing memory and the degree of hearing drop is evidence of the possible influence of functional noise on this condition. The absence of a similar connection in all the groups with a service life up to 10 years (see Table ), as well as a percent which is similarly high independent of the persons with poor memory, allow us to assume that the drop in the hearing memory sets in with workers under noisy conditions (during the first year of work), sooner than the hearing decline (after 3-5 years).

The visual memory and the attention concentration at the stage up to the 10th year was seldom lower in the case of workers with nerve tension labor (in 8-11% of the cases), but in 20-53% of the cases, the drop of these functions was observed in people with physical labor. Evidently, the determined influence here showed an elemental professional selection of the workers. The very character of the labor of the workers of the 2nd group required good memory and attention, which is important even with physical labor. A decline of such functions of high nerve
activity such as memory and attention concentration may be related to the number of adequate reactions of the organism to the effect of intense noise.

**Literature**


Received March 14, 1975
Кандидаты наук Г. С. Зверева и М. В. Раппинер, А. В. Комиссаров

ШУМ ПРОКАТНОГО ПРОИЗВОДСТВА И ЕГО ВЛИЯНИЕ НА ОРГАНИЗМ РАБОЧИХ

Научно-исследовательский институт техники труда и профзаболеваний, Донецк

По наблюдениям ряд авторов, влияние на организм человека промышленного шума отличается от действия стационарного шума. Некоторые физиологические и системы исследованиями установлено, что шум, выделяемый промышленным оборудованием в период его работы, оказывает более интенсивное воздействие на организм человека, чем шум, выделяемый при обычных условиях эксплуатации.

В трубопроводных системах на участках непрерывного технологического процесса, особенно в процессах нагревания металла, шум может достигать значительных уровней. Вследствие этого, в ряде случаев, необходимо проводить специальные исследования, направленные на оценку шума и его влияния на организм человека.

Влияние производственного шума и других шумов на организм человека

<table>
<thead>
<tr>
<th>Вид шума</th>
<th>Состояние функции памяти и внимания</th>
</tr>
</thead>
<tbody>
<tr>
<td>Шумы</td>
<td>до 10</td>
</tr>
<tr>
<td>Шумы</td>
<td>10-15</td>
</tr>
<tr>
<td>Шумы</td>
<td>15-20</td>
</tr>
<tr>
<td>Шумы</td>
<td>20-25</td>
</tr>
</tbody>
</table>

Влияние шума на функции на организм

<table>
<thead>
<tr>
<th>Вид шума</th>
<th>Физиологическое действие</th>
</tr>
</thead>
<tbody>
<tr>
<td>Шумы</td>
<td>0-5</td>
</tr>
<tr>
<td>Шумы</td>
<td>5-10</td>
</tr>
<tr>
<td>Шумы</td>
<td>10-15</td>
</tr>
<tr>
<td>Шумы</td>
<td>15-20</td>
</tr>
</tbody>
</table>

Приемы и методы измерения шума:

1. Амплитудно-частотный спектр
2. Спектральная плотность
3. Спектральная и гармоническая спектроскопия
4. Моменты спектра
5. Спектральные характеристики

Таким образом, шум, выделяемый в процессе производства, оказывает значительное влияние на организм человека, вызывая различного рода нарушения функций и органов.

### Таблица 1

<table>
<thead>
<tr>
<th>Состояние функции памяти и внимания</th>
<th>Стаж работы (в годах)</th>
<th>Коэффициент резко выраженной ( r )-отрицательной связи при стаже до 10 лет</th>
<th>более 10 лет</th>
</tr>
</thead>
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<tr>
<td></td>
<td>до 1</td>
<td>1—3</td>
<td>3—5</td>
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<tr>
<td>Снижение памяти: слуховой</td>
<td>41,7</td>
<td>61,1</td>
<td>40,7</td>
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<tr>
<td></td>
<td>3,4</td>
<td>1,1</td>
<td>14,8</td>
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<td>Снижение концентрации внимания</td>
<td>16,7</td>
<td>22,1</td>
<td>35,3</td>
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<td>9,5</td>
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### Таблица 2

<table>
<thead>
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<th>Состояние функции памяти и внимания</th>
<th>Физический</th>
<th>Нервно-сосудистый</th>
<th>Неврологический</th>
<th>Не выражен</th>
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<td>37,7</td>
<td>57,9</td>
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<td>11,5</td>
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<tr>
<td></td>
<td>39,0</td>
<td>54,8</td>
<td>11,5</td>
<td>69,5</td>
</tr>
</tbody>
</table>

**Примечание:** знак * при цифре — наличие прямой тесной корреляционной зависимости от снижения слуха (коэффициент корреляции \( r \) > 0,5).

Таблицы, схемы, в том числе и векторные, включены в текст. Отсутствует описание графиков и диаграмм. В тексте содержатся ссылки на литературные источники.

### Литература


Поступила 15 III 1974 г.
### Summary Form for Studies on the Effects of Noise on the Cardiovascular System (CVS)

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tbody>
<tr>
<td>Y. Ando (Dritte Physikalisches Institut, Universität Goettingen, Federal Republic of Germany); H. Nattori</td>
<td>Kobe University Hospital, Ikuta, Kobe, Japan</td>
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</tbody>
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<th>Investigator's Phone No.</th>
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### Citation

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<th># of Fig.'s</th>
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### Purpose of study
To test for significant differences in maternal HPL levels in women living in noisy or quiet areas.

### Type & duration of experiment
Short-term epidemiological survey using blood samples taken at same times from both test and control groups.

### Description of test groups (subject numbers)
2 groups: (1) test—In the Itami district near Osaka International Airport (noisy area)—143 randomly selected pregnant women 22-41 weeks gestation
(2) controls—Kobe district (little aircraft noise)—112 randomly selected pregnant women, 22-41 weeks gestation

### Control of other stressors
Environmental conditions similar for test & control areas (air pollution, pop. density, weather, traffic, standard of living).

### Noise Stimulus
Source: Aircraft noise (people tested who lived near a large airport).
Spectral characteristics: Not given.
Noise levels: WEPNL (weighted equiv. continuous perceived noise level) 75-95
Length of exposure: Variable.

### # of trials
1 sampling per subject.

### Statistical Methods
Chi-square two-sided test.

### CVS Response Measured
None.

### Nonauditory effects
Human Prenatal Lactogen (HPL) levels measured in maternal plasma in both noisy and quiet groups: HPL levels lower after 30th week of pregnancy in noise-exposed groups; significant difference after 35th week.

### Author's conclusion
Human placental lactogen (HPL) levels of subjects in the high aircraft noise area were lower than those in the control area after the 30th week of gestation. The percentage of HPL levels in the fetal danger range (4 micrograms/mL or less) was higher in the noise area. Lower birth weights of babies were associated with lower HPL levels in the noise area.

### Evaluation & comments
1. Interesting implications for potential hazards of noise on human reproduction
2. It was not clear from the article whether HFL levels were tested in serum or plasma, since the terms were used interchangeably.

Summary form only
EFFECTS OF NOISE ON HUMAN PLACENTAL LACTOGEN (HPL) LEVELS IN MATERNAL PLASMA

BY

Y. ANDO*
Faculty of Engineering, Kobe University, Nada, Kobe, Japan 657

AND

H. HATTORI
Faculty of Medicine, Kobe University, Ikuta, Kobe, Japan 650

Summary

The levels of human placental lactogen (HPL) in the serum of expectant mothers both subjected to and not subjected to aircraft noise were measured. The HPL levels in the quiet reference area and in the noise area were similar before the 29th week of pregnancy. However, the HPL levels of subjects in the noise area tended to be lower than those in the reference area after the 30th week of pregnancy and the difference became significant after the 36th week of pregnancy. The lower HPL levels were associated with lower birth weight for infants of mothers who lived in the noise area.

The manner in which babies react to aircraft noise during their sleep as a function of the length of stay in a noisy area has been investigated by a statistical survey (Ando and Hattori, 1970, 1973 and 1974). It was found that babies born to mothers who moved to the noise area before or during the first five months of pregnancy showed little or no reaction to the noise. After the introduction of regular jet plane services, it was observed that the average birth weight of babies in the noise area (which surrounded an international airport) was clearly less than that from other neighbouring quiet areas. The incidence of low birth weight babies increased as the noise level increased. It has been suggested that such noise could be a possible cause of toxemia of pregnancy (Ando and Hattori, 1974).

We now report HPL levels in maternal serum in a noise area and a quiet reference area.

METHODS

In the Itami district around the Osaka International Airport (hereafter referred to as the noise area), blood samples were taken from 343 randomly selected expectant mothers, between 22 and 41 weeks gestation, who were attending the Itami City Hospital. In the Kobe district, a district with relatively little aircraft noise (hereafter referred to as the reference area), blood samples were obtained from 112 randomly selected expectant mothers of equivalent gestation who were attending either the Kobe University Hospital or the Kaneko Hospital. Other environmental conditions such as atmosphere pollution, density of population, standard of living, weather and traffic conditions were almost the same for the two areas. The noise area was residential and commercial while the reference area was only commercial and industrial.

One 5 ml sample of venous blood was taken from each subject and refrigerated overnight. On the following day about 2 ml of serum was
separated from each sample and then stored in a deep freezer. The HPL levels were measured at Kobe University Hospital. The measurements were conducted a total of five times with 50 to 170 samples being processed in one batch. The measurements were made using the Phadebas HCS Test Kit (Lot No. 1087, 6121 and 5546). As far as possible, samples from mothers in the noise area and mothers in the reference area were measured at the same time. At the time of blood sampling, the names of the mothers, the date of sampling, and the estimated date of delivery were recorded. In the Isami district, the time at which the mothers had moved into the noise area and their current addresses were also recorded. The birth weights of the babies were obtained from hospital records.

RESULTS

Figures 1 and 2 show the HPL levels in mothers in the reference area at varying stages of gestation. The graphs also show the mean (+2SD) levels (Lindberg and Nilsson, 1973a) and the fetal danger zone (FD Zone) is outlined. It is apparent that most of the HPL levels of mothers in the reference area remained within the normal range, while HPL levels of mothers in the noise area, where the noise levels were in the range of 75 to 95 WEPNL (see Appendix), were lower, particularly after the 36th week of pregnancy.

The total number of subjects and the percentage of subjects whose HPL levels were more than 1SD below the mean, from both the noise area and the reference area, are shown in Table 1.

The incidence of HPL levels more than 1SD below the mean at various stages of pregnancy is shown in Figure 3. Table 1 and Figure 3 show that after the 32nd week of pregnancy, the HPL levels of more than 40 per cent of the subjects in the noise area had fallen to more than 1SD below the mean and that this percentage was higher for subjects at later stages of pregnancy. Significant differences were obtained after the 36th week of pregnancy (Chi square two-sided test; P<0.025).

The correlation between the HPL level measured during the 30th to 40th weeks of pregnancy and the birth weight of the babies in the noise area was also examined. The mean (+1SD) birth weight of babies born to mothers whose HPL levels were 5 mg/ml or above was 3307±359 g (39 subjects). On the other hand, the mean (+1SD) birth weight of babies born to mothers with HPL levels of 4 mg/ml or less was 2945±263 g (11 subjects). In these two groups, the incidence of birth weights below 3.0 kg was 77 per cent (HPL levels 6 mg/ml or above and 73 per cent (HPL levels 4 mg/ml or
**TABLE I**

Percentage of subjects whose HPL levels were more than 1SD below the means

<table>
<thead>
<tr>
<th>Gestation (weeks)</th>
<th>No. of subjects in reference area</th>
<th>No. of subjects in noise area</th>
<th>P value for difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>With HPL levels more than 1SD below mean</td>
<td>Rate (per cent)</td>
</tr>
<tr>
<td>22-23</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>24-25</td>
<td>6</td>
<td>2</td>
<td>40</td>
</tr>
<tr>
<td>26-27</td>
<td>5</td>
<td>2</td>
<td>40</td>
</tr>
<tr>
<td>28-29</td>
<td>13</td>
<td>3</td>
<td>20</td>
</tr>
<tr>
<td>30-31</td>
<td>11</td>
<td>2</td>
<td>18</td>
</tr>
<tr>
<td>32-33</td>
<td>16</td>
<td>4</td>
<td>25</td>
</tr>
<tr>
<td>34-35</td>
<td>13</td>
<td>3</td>
<td>23</td>
</tr>
<tr>
<td>36-37</td>
<td>17</td>
<td>4</td>
<td>23</td>
</tr>
<tr>
<td>38-39</td>
<td>14</td>
<td>4</td>
<td>29</td>
</tr>
<tr>
<td>40-41</td>
<td>12</td>
<td>3</td>
<td>25</td>
</tr>
<tr>
<td>Total</td>
<td>112</td>
<td>25</td>
<td>22</td>
</tr>
</tbody>
</table>

**TABLE II**

Percentage of subjects categorized according to HPL level

<table>
<thead>
<tr>
<th>Category</th>
<th>Reference area</th>
<th>Noise-area</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of Subjects</td>
<td>Rate (per cent)</td>
</tr>
<tr>
<td>(a) 30th to 35th week of pregnancy</td>
<td>4 μg/ml or less</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>Below the lower range of normal HPL level (−2SD)</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>6 μg/ml or more</td>
<td>40</td>
</tr>
<tr>
<td>(b) 36th to 41st week of pregnancy</td>
<td>4 μg/ml or less</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>Below the lower range of normal HPL level (−2SD)</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>6 μg/ml or more</td>
<td>43</td>
</tr>
</tbody>
</table>

All results obtained at 36 to 41 weeks were significantly different (P<0.01)

less) respectively; the difference was significant (P<0.005).

**DISCUSSION**

This study shows that the HPL levels of subjects in the noise area tended to be lower than those in the reference area. The difference was particularly distinctive for the HPL levels measured after the 36th week of pregnancy. Furthermore, the percentage of mothers with HPL levels of 4 μg/ml or less, a measure of fetal danger, tended to be higher in the noise area than in the reference area.

We also confirmed that the birth weight of babies born to mothers with low HPL levels tended to be low (Letchworth et al, 1971; Spalding et al, 1971; Lindberg and Nilsson, 1973b). The decrease of the HPL level was associated with a lower birth weight for babies whose mothers lived in the noise area and this
The percentage of subjects with HPL levels more than 18D below the mean by stage of pregnancy (see Table I), confirmed previous results (Ando and Hattori, 1973 and 1974).

ACKNOWLEDGEMENTS

The authors are grateful to Dr Himi of the Kaneko Hospital in Kobe for taking blood samples and measuring HPL levels and to Dr Kasuma of the Hyogo City Hospital for taking blood samples. They thank Dr Mochizuki and Dr Morikawa, Faculty of Medicine, Kobe University for helpful discussions; the patients for their co-operation and M. Shobatake for help with translation. This research was supported by the Ministry of Education, Japan.

APPENDIX

The weighted equivalent continuous perceived noise level (WECPNL) is calculated as follows.

\[ \text{WECPNL} = \frac{d(B(A)-10 \log N-27}{\text{REFERENCES}} \]

where \( d(B(A) \) is the average value of peak noise level in \( d(B(A) \) and \( N \) is the number of flights in a day, i.e. \( N = N_1 + 3N_2 + 10N_3 + N_4 \), \( N_1 \), \( N_2 \), and \( N_4 \) are the number of flights between 0700 and 1859 hours, 1900 and 2159 hours and 2200 and 0659 hours respectively. WECPNL was nearly equal to 2 + ECPNL for the distribution of noise level in ECPNL (see Ando and Hattori, 1973).

REFERENCES

### STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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</thead>
<tbody>
<tr>
<td>Edwin Boyle, M.D.</td>
<td>Miami Heart Institute Research Division</td>
</tr>
<tr>
<td></td>
<td>4701 N. Meridian Ave. Miami Beach, Fla. 33140</td>
</tr>
<tr>
<td>Phillip A. Villanueva</td>
<td></td>
</tr>
</tbody>
</table>

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#### Citation

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<thead>
<tr>
<th>Type &amp; Duration of Experiment</th>
<th>Purpose for Study</th>
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<tbody>
<tr>
<td>Incidence of seiures with various handling procedures and hyperbaric oxygen flow rates for laboratory rats were compared.</td>
<td>To test whether noise and handling of animals were significant stressors contributing to HBO-induced seiures.</td>
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</table>

<table>
<thead>
<tr>
<th>Description of Test Groups (Subjects, Age, Sex, etc.)</th>
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</thead>
<tbody>
<tr>
<td>Test rats: hyperbaric air of 5000 ml/sec which was reduced to 30 ml/sec and normal rough handling; 30 ml/sec air flow and gentle handling Control rats: normobaric air in similar conditions as tests</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Control of Other Stressors</th>
<th>Statistical Methods</th>
</tr>
</thead>
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<tr>
<td>controlled laboratory conditions</td>
<td>not given--mostly a qualitative study</td>
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<table>
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<tr>
<th>Noise Stimulus</th>
<th>CVS Response Measured</th>
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<tr>
<td>source: flow of oxygen in HBO chamber (loud hiss)</td>
<td></td>
</tr>
<tr>
<td>Spectral characteristics: not known</td>
<td></td>
</tr>
<tr>
<td>noise level: varied with flow rate of oxygen</td>
<td></td>
</tr>
<tr>
<td>length of exposure: 60 min.</td>
<td></td>
</tr>
<tr>
<td>0 of trials: not given specifically, but at least 30 treatments of noise &amp; handling methods</td>
<td></td>
</tr>
</tbody>
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Author's conclusions: A combination of rough handling and loud gas flow noise in a hyperbaric oxygen chamber is a significant contributor to hyperbaric oxygen (HBO)-induced seiures in rats. The seiures may be mediated by an adrenocortical mechanism.

**Evaluation & Comments**

(1) This study may be mostly of use to laboratory animal caretakers; (2) shows another aspect of noise as a stressor; (3) effects of noise and handling were not studied separately.

Summary form only
HYPERBARIC OXYGEN SEIZURES IN RATS: EFFECTS OF HANDLING AND CHAMBER NOISE.

EDWIN BOYLE AND PHILIP A. VILLANUEVA

The convulsive effects of hyperbaric oxygen (HBO) were first reported nearly a century ago (1) and have been reported under various conditions to be considered a true complication of exposure to oxygen under high pressure. Susceptibility of animals, and in particular, HBO-induced seizures and to oxygen toxicity in general, may depend upon such factors as age (2), strain of rat (3), and temperature (4).

Due to these seizures, other effects of HBO have become difficult to analyze. Part of our group's study of the effects of HBO upon sensory function in humans has been an examination of the effects of HBO upon neurotransmitters in rat brains. However, previous studies of the effects of HBO upon neurotransmitters in rats and mice have reported oxygen-induced seizures (5,6).

In one of our early studies, using male rats (Can: COBS [V7]: III) from the Charles River Labs ranging in age from 24-26 mm of age, we noted frequent seizures when the animals were exposed to HBO at 50 psig. The animals were exposed for a total of 60 min (15 min for both compression and decompression, allowing a sustained 50 psig exposure of 30 min). Control rats exposed to normobaric air in similar chambers and at the same schedule showed no such convulsions. After 5 exposures we noted that the rats had become less responsive to stimuli and had a tendency to be aggressive, even when the chamber was not pressurized.

The flow rate of oxygen was then reduced to 50 ml/min. The occurrence of seizures was reduced in the test animals but not eliminated. It was therefore concluded that the seizures were not due to the high oxygen concentration but rather to the high pressure itself. Further studies are needed to determine the exact mechanism of these seizures and their relationship to other effects of HBO.

REFERENCES
use in one group of rats at the same time. The results, summarized in Table 1, show that the rats in Group A, which were administered the drug, had a significant increase in heart rate compared to the controls (Group B) that were not given the drug. The differences in heart rate were statistically significant (p < 0.05). These findings suggest that the drug may have a positive effect on heart rate in these rats. Further studies are needed to investigate the mechanism of action and potential therapeutic applications of this drug.
**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tr>
<td>Robert W. Contrell</td>
<td>NA</td>
</tr>
<tr>
<td>Captain, Medical Corps, U.S. Navy</td>
<td>Naval Regional Med. Ctr.; San Diego, CA 92134</td>
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<th>Sponsoring Organization</th>
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**Description of test groups (subjects, age, etc.):**
NA

**Control of other variables:**
NA

**Noise Stimulus:**
NA

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<th>NA</th>
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</table>

**Source:**
NA

**Spectral characteristics:**
Non-auditory effects
peripheral vasodilation and other circulatory changes changes in gastrointestinal motility change in Galvanic Skin Response increased corticosterone levels increased cholesterol levels increased fat, portions and birth defects increased catecholamine secretion

**Evaluative comments:**
A very informative review of some of the major areas of the non-auditory physiological effects of noise; includes a table listing the functions of the autonomic nervous system (sympathetic and parasympathetic nerves) which is considered the mediator of physiological responses to noise. Theoretical mechanisms involved in the stress reaction to noise are discussed.

**Author's conclusion:**
The physiological effects of noise are more dangerous than is generally believed. More study is needed.
SUMMARY

It is well known that noise adversely affects mankind. Many studies have been performed which show the effects of noise on hearing, speech reception, sleep, mental health and work performance. Until recently, relatively little had been written on the physiological effects of noise. Studies which have been reported are generally retrospective on a group of people working in a noisy environment where precise controls over the intensity and the duration of exposure were lacking. Recent studies show that the effect of noise on the biochemistry of the body, the cardiovascular system, and the organ systems controlled by the autonomic nervous system are more serious than previously suspected.

Noise serves as a stressful stimulus which provokes the General Adaptation Syndrome as described by Selye. Noise is one of the serious stressful stimuli which the body attempts to counteract via the hypothalamus to the pituitary which produces ACTH resulting in increased adrenocortical activity. There is considerable evidence to support this concept, and this theory along with recently carefully controlled studies, are reviewed.

INTRODUCTION

Noise has been recognized as harmful to man for at least 2500 years. The Sybarites of Greece banned metalwark involving hammering within the city limits as early as 400 B.C. As industrialization and the urbanization of population density, noise levels have risen to a point where they have become a serious public health problem.

The effects of noise on hearing, annoyance, sleep and speech interference, and work performance have been studied extensively. Until recently relatively little has been written on the physiological effects of noise. Most of the work has been done as retrospective analysis on groups of people working in a noisy environment where precise measures of the intensity of the noise and the duration of exposure were lacking. In the studies, it was not always possible to separate the effects of heat, light, dust, radiation from other environmental pollutants from the effects of noise alone.

Recent studies show that the effect of noise on the biochemistry of the body, the cardiovascular system, and the organ systems controlled by the autonomic nervous system are more serious than previously suspected.

In Germany, Jansen, Lehmann, and Meyer-Dollies; in Russia, Andriukhin, Andrujkovitch, and Shatalov; among others; and in the U.S. Davis, Rosen and more recently Central have studied the physiological effects of noise exposure. A symposium held in Boston in 1969 was devoted to this subject, and resulted in a book which detailed much of the knowledge available at that time.

PATHWAYS

A pathway from the sound source to the target organ must be established in order to show a cause-and-effect relationship. For the purposes of this discussion, infrasound (below 20 Hz) and ultrasound (above 20,000 Hz) will not be considered. Nor will the possible physiological effects of vibratory energy on the body in general be considered. There is evidence that vibratory energy can affect the body if the vibrations transmitted through structures is more significant than airborne transmission and the vibrations (sound) from 20 to 20,000 Hz may exert a physiological effect in ways other than through the auditory mechanism, i.e through the skin. These studies are scarce and since the most damaging of vibratory (noise) effects are transmitted through the ear with its central auditory connections, it is those physiological effects which will be considered here.

Once sound enters the auditory canal, it causes the tympanic membrane to vibrate. This in turn moves the three ossicles which at the oval window create a wave in the inner ear. Collisions of the footplate of the stapedial footplate of the tympanic membrane with the small area of the stapedial footplate (1/1200) plus a small lever advantage from the ossicles, transform the small pressure of sound energy impinging on the tympanic membrane into a 25-fold greater force acting on the inner ear fluid. The fluid wave thus created distorts the basilar membrane and the hair cells of the organ of Corti are stimulated. Nerve impulses generated in the organ of Corti travel along the auditory nerves to the central auditory nuclei.

Sound is also transmitted through the bones of the skull directly to the inner ear, and we measure these pathways (air conducted and bone conducted sounds) to help diagnose hearing disorders. One who cannot hear air conducted sound but can hear by
bone conduction has a conductive hearing loss which in most cases can be remedied by
appropriate medical or surgical treatment. If one has difficulty hearing both air and
bone conduction sound, this is known as nerve deafness which is not correctable, and
sound must be amplified in order for the patient to hear.

After stimulating the auditory nerve, the sound waves, which are now nervous im-
ulses, travel to the cental auditory nuclei in the medulla, where some fibers ascend
through the midbrain via the lateral lemniscus on the same side, but some cross before
ascending on the opposite side through the midbrain to reach the inferior colliculus,
then the medial geniculate body and finally the auditory area of the temporal lobe
where the sound is interpreted.

It is probable that after reaching the central auditory nuclei, impulses travel
through the reticular formation to reach the hypothalamic nuclei. From the hypothalamic
nuclei which are situated just superior to the pituitary, the products of stimulation
travel to the pituitary which then produces endocrine effects and completes the auditory-
hypothalamic-pituitary-endocrine pathway.

The hypothalamus is not the only part of the brain directing autonomic activity.
The forebrain, the thalamus and the cerebral cortex are all integrated with the hypo-
thalamic to utilize behavioral and autonomic adjustments which serve to adapt the indi-
vidual to changes in both the internal and external environment.

AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system (ANS), also known as the vegetative nervous system, is
a system of motor neurons whose cell bodies are collected into ganglionic chains in the
thoracic region near the vertebral column and in isolated ganglia elsewhere in the
body. Anatomy of the ANS is divided into the thoracolumbar (sympathetic) and cranio-
scotral (parasympathetic) division. This system is generally not under voluntary control.

Table I lists the functions of the autonomic nervous system, which acts to maintain
the constancy (homeostasis) of the fluid environment (internal milieu) of the body. The
autonomic nervous system combats forces which tend to cause variations in this environment.
It regulates the composition of body fluids, their temperature, quantity and distribution
by affecting changes in circulatory, respiratory, excretory and glandular organs.

**TABLE 1**

<table>
<thead>
<tr>
<th>AUTONOMIC NERVOUS SYSTEM FUNCTION</th>
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<tbody>
<tr>
<td><strong>SYSTEM</strong></td>
</tr>
<tr>
<td>Pupil (Pupil)</td>
</tr>
<tr>
<td>Heart Rate</td>
</tr>
<tr>
<td>Blood Vessels</td>
</tr>
<tr>
<td>Coronary</td>
</tr>
<tr>
<td>Skin &amp; Mucosa</td>
</tr>
<tr>
<td>Skeletal Muscle</td>
</tr>
<tr>
<td>Carotid</td>
</tr>
<tr>
<td>Pulmonary</td>
</tr>
<tr>
<td>Abdominal Viscera</td>
</tr>
<tr>
<td>Bladder</td>
</tr>
<tr>
<td>Glande</td>
</tr>
<tr>
<td>Sweat</td>
</tr>
<tr>
<td>Salivary</td>
</tr>
<tr>
<td>Gastric</td>
</tr>
<tr>
<td>Adrenal</td>
</tr>
<tr>
<td>Smooth Muscle</td>
</tr>
<tr>
<td>Skin (Pilomotor)</td>
</tr>
<tr>
<td>Stomach</td>
</tr>
<tr>
<td>Small &amp; Large</td>
</tr>
<tr>
<td>Intestine</td>
</tr>
<tr>
<td>Bladder</td>
</tr>
<tr>
<td>General</td>
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<td>Mediatar</td>
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</table>

The two divisions are antagonistic: one slows, the other speeds the cardiac rate; one constricts, the other dilates the pupil or the bronchi. Generally, the sympathetic strengthens the defense against such challenges as enemy attacks, temperature variations, and water deprivation. Animals who have had sympathectomies are incapable of working (no sugar is mobilized); cannot withstand exposure to temperature extremes (no sweating when hot; no vasoconstriction when cold) and they are less able than normals to withstand oxygen deprivation or hemorrhage. They can survive in a controlled environment.

The parasympathetic system is concerned with restoration of the body processes. It
inhibits the heart rate, contracts the pupils and stimulates the digestive tract through
which the energy stores of the body are replenished. It is primarily in control while one
is sleeping.
DISCUSSION

Selyes described the General Adaptation Syndrome. According to this concept, an individual exposed to stress - cold, heat, drug reaction, fractures, infections, operations, burns or other trauma (NOISE) - responds by:

1. Stimulation of the hypothalamus which
2. Stimulates the anterior pituitary to release ACTH which
3. Stimulates the adrenal cortex to release cortisol which
4. Stimulates the body to protect against systemic anabolism of tissue.

This theory is well accepted even if there is not complete agreement as to the actual mechanism of action. Stress is known to be a factor in the development of such diseases as peptic ulcers, cardiovascular disease, including hypertension and coronary artery disease, and it is implicated in the aging process.

Noise, especially aversive, intrusive noise, is thought to be merely one of many agents which serves as a stress-provoking stimulus. Noise stimulates the sympathetic portion of the ANS. As such it should be minimized just as noxious fumes, excessive heat or cold or, indeed, even marked population density, should be diminished and controlled where possible in the environment.

Assuming this to be true, what evidence do we have that noise has any effect on these functions?

Mason reported the electrical stimulation of the hypothalamus of conscious rhesus monkeys was associated with an increase in pituitary-adrenal-cortical activity, as judged by the marked elevation of plasma 11-hydroxycorticoesteroid.

![Graph showing circulatory reaction during noise exposure](image-url)
### PERIPHERAL CIRCULATION PROBLEMS

<table>
<thead>
<tr>
<th>N = 410</th>
<th>VERY NOISY INDUSTRIES</th>
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<tr>
<td>N = 165</td>
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<th>HEART PROBLEMS</th>
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<table>
<thead>
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<th>EQUILIBRIUM DISTURBANCE</th>
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<tbody>
<tr>
<td>N = 128</td>
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<td>N = 51</td>
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#### FIG. 2
Differences in percentages of occurrence of physiological problems in 1,605 German industrial workers. The differences in peripheral circulation and heart problems in the two classes of industry were statistically significant. (From G. Jansen, Stahl, Eisen, 1951, pp. 237-238. With permission of the author and Stahl, Eisen.)

Henkin and Knigge exposed rats to 130 dB at 220 Hz for 48 hours and measured adrenal secretion of corticosterone. It was found that the output of adrenal corticosterone doubled in 30 minutes and tripled in 60 minutes. This tripled secretion rate was maintained for approximately 12 hours at which time it fell to normal or subnormal levels only to rise again to the former high rate where it was maintained for 25 to 48 hours.

Friedman et al. exposed rats to 102 dB of white noise for 10 weeks and showed a much higher level of blood cholesterol than non-exposed animals despite being on identical diets. The animals exposed to noise developed more aortic atherosclerosis and more cholesterol deposits in the arteries than the control animals.

Weber exposed gravid female rats to noise intensities of 75-94 dB from 20 to 1,500 Hz for 6 minutes of each hour followed by 54 minutes of quiet (ambient noise level was 64 dB) and to a flashing light for the same period of time, throughout each day of pregnancy or to some other desired day (i.e. 15-20 days).

He found:

1. Total litter resorption occurred in 40-50 percent of the pregnancies.
2. Increased congenital anomalies, including meningocoeles, spinal bifida, cranial hernias, abdominal hernias, and defects of the eye, tail, hind- and forefoot.

Gebert and Anderson studied the effects of chronic intermittent noise stress on the body weight and the weight of the ventricles, adrenal glands, kidneys, and ovaries of young and old rats and rabbits. Significantly hypertrophied ventricles of both species were found at the end of three weeks' stress. Body and other organ weights were slightly decreased, with the exception of the adrenals and kidneys of the older rats which were increased.
FIG. 1. Incidence of hypertension in male and female workers (in age groups under and above 40 years) in noisy workshops: 1. toolmaking workshop; 2. sorting workshop; 3. workshop with automatic lathes; and 4. workshop producing ball bearings. (From A. A. Andriukhin, Cor. Vasea., 1961, pp. 205-209. With permission).

Similar evidence is available in humans. Davis et al.10 labeled the following set of responses to noise the N-response:

1. A vascular response characterized by peripheral vasoconstriction, minor changes in heart rate, and increased cerebral blood flow since cerebral vessels show no vasoconstriction to such stimuli.

2. Slow, deep breathing.

3. A change in the resistance of the skin to electricity (Galvanic skin response [G.S.R.]).

4. A brief change in skeletal muscle tension.

To this set of responses can be added:

1. Changes in gastrointestinal motility.

2. Chemical changes in blood and urine from endocrine glandular stimulation.

A tone of approximately 70 db SPL at 1,000 Hz will elicit the N-response. This same tone continued for a long enough time may induce TS or NIPTS and is near the level at which broadband noises may become significantly aversive to people.10

Davis and Berry10 and Stern21 found that humans who could avoid an 80 db, 10-second 100 Hz tone by pushing a switch at the correct time, exhibited greater gastro-intestinal motility during the tone (i.e., when they failed to press the switch) than did subjects who had no means of avoiding the tone. Kryter18 labeled this a response-contingent effect of noise. The noise thus becomes an aversive stimulus, primarily because it
indicated incorrect responses on the part of the subject; its aversive effect otherwise was small.

Hornmann, et al.22 in a similar study verified the aversive effects of noise with three groups of subjects exposed to white noise at 95 dB. For Group A, the noise signaled they had made an error in a pseudo-tracking task; for Group B the same noise was the signal that they were on target in the pseudo-tracking task; and for Group C the noise was heard without any task.

Measures were: (a) TTS; (b) muscle tension measured by electromyography; and (c) subjective scaling of the amount of annoyance and disturbance induced by the noise and of the general sensitivity to noise, of the subjects.

The results showed subjects who invest the noise with positive emotional valence, feel themselves less annoyed, less disturbed and, in general, less susceptible to the noise than subjects who receive the noise with negative valence.

Muscle tension was highest for Group A, less for Group B, and least for Group C.

The amount of TTS was dependent upon the valence of the noise:

1. Negative valence (Group A): TTS=10.1 dB.
2. Positive valence (Group B): TTS=12.0 dB.
3. Neutral valence (Group C): TTS=11.0 dB.

The response-contingent effect apparently does not hold for all physiological reactions to noise. Jansen and Kleinert2 found similar responses in the circulatory system (cardiac output, minute flow volume) in subjects exposed to random noise or music of equal intensity (about 90 Phn). Although the cardiac output and minute flow volume increased in some subjects and decreased in others, indicating an individual difference in

Somatic responses to sound, it was the intensity of the sound and not its aversive (noise) or pleasurable (music) aspect which controlled somatic responses.

Levi24 measured urinary catecholamines as a method of determining 8-responses in human subjects. He found the following:

1. Pleasant stimuli (motion pictures evoking amusement) were nearly as potent as unpleasant stimuli (motion pictures evoking anger) in causing increased excretion of catecholamines.

2. Work in industrial noise and office work caused increased excretion of catecholamines.

3. Noise, light, or task have less influence on the catecholamine excretion levels than does the subject's attitude.

4. Under experimental stress, emotionally vulnerable people as a group do not excrete more catecholamines than normal people.

Lehmann and Tamm25 studied circulatory changes in human subjects exposed to noise. Peripheral arterial resistance was found to increase under the effect of noise, but pulse frequency and blood pressure remained unaffected. FIG. 1. summarizes the circulatory reactions observed by Lehmann and Tamm.

Meyer-Delius26 related the circulatory effects to the duration of noise exposure. An exposure of 90 dB(A) for 20 seconds increased peripheral arterial resistance for 80 seconds, i.e. the vasodilatation mediated through the autonomic nervous system in response to noise exposure persists after the exposure.

There is a physiological adaptation to sound. Habituation might be a more correct term, but adaptation is used more frequently in the literature when referring to this phenomena. Bartos, et al. found that acceleration of the heart rate in newborn babies exposed to bursts of acoustic clicks at 85 db adapted by the end of 40 trials. This adaptation is not complete and obviously does not cover all N-responses. Although man adapts to background noise, he will respond when the character or intensity of the noise is changed. Rossi, et al.26, found adaptation of vasaconstriction in subjects exposed to a background noise of 70 db at 500 Hz did not reduce vasoconstriction to superimposed tones of 60 to 105 db at 2000 Hz.

Jansen27 plotted the increased numbers of peripheral circulatory problems, heart problems and equilibrium disturbances in German industrial workers in very noisy industries. The differences in peripheral circulation and cardiac problems in the two groups were statistically significant (FIG. 2).

Andriuki2 showed a greater incidence of hypertension in men and women working in very noisy areas than those working in less noisy areas (FIG. 3). There was also a relationship to age with older people appearing to exhibit more hypertension.

Additional data from Russia2,8 has shown that among workers in industries with high noise levels there is an unusually high incidence of circulatory, digestive, metabolic, neurologic and psychiatric problems.

Rosen and co-workers7 studied the Nubians, a primitive tribe living in the Sudan. This tribe has no firearms or drums; their diet consists mainly of vegetables and some fish; and they live in very quiet surroundings (35-40 db[C]) with relatively little stress. Hearing loss, hypertension and cardiovascular disease is virtually unknown even into old age.
Once the inhabitants move to noisy Karloura, where they are exposed to noise, stress and a diet similar to city dwellers, they are reported to develop hypertension, coronary artery disease and hearing loss.

Critics of the concept that noise can cause pronounced physiological effects point out that a given noise exposure does not affect all persons similarly, nor does the same individual respond similarly to a given noise exposure occurring at a different time. These are valid observations which complicate the investigation of noise effects. A partially satisfactory answer is that no two humans are alike nor do they respond identically to any stress-stimuli. Normann's study of the valence effect of noise (i.e., a desired sound is less damaging than an unwanted sound) is an interesting concept. This may give some insight into why some people are content to work in a noisy stimulus and show little or no effects, whereas others are upset by the same noxious stimulus and respond with the symptoms of stress.

A very interesting experiment is reported by Jansen. He measured the change in finger pulse amplitude from pre-emissions to transmittance of noise at 90 dB(A) 20 seconds after the onset of white noise at 85 dB(A) and 9 minutes after the onset of white noise at 105 dB(A). Hearing was measured before exposure and the TTS at 2 minutes after exposure ceased. The TTS was measured at 4000 Hz. Fig. 4 is a graph relating TTS and change in finger pulse amplitude (peripheral vasoconstriction). Note that for short exposures wherein you would expect little or no TTS there was rather greatly decreased PPA. With longer exposures the TTS and change in PPA were similar. Jansen concluded that the vegetative response (VHR), as manifested by changes in the finger pulse amplitude, and TTS can be influenced by noise acting through the vegetative system (A.M.E.). Furthermore, a man who has not have a hearing loss from high intensity noises is, nevertheless, highly endangered by the non-auditory physiological effects of high intensity noise.

In studies of our own, we noted that even though 20 healthy young subjects showed little (1-8 dB) TTS after 10 days exposure to intermittent noise presented 0.66 seconds every 22 seconds 24 hours per day, they did have statistically significant shifts in plasma cortisol levels (Fig. 5) and blood cholesterol levels (Fig. 6). This noise was in the 4000-6000 Hz range and was presented at 85, 85 and 90 dB(A) each for 10 days. These are allowable levels by many damage risk criteria. This stimulus caused reduction in finger pulse amplitude during sleep and this, coupled with the relatively small TTS, supports Jansen's findings.

The shift in the blood cholesterol and plasma cortisol levels is interesting. Plasma cortisol is known to be influenced by ACTH and other studies have suggested that stress will elevate cholesterol and cortisol. Although controversy exists as to the normal values for serum cholesterol, the range is roughly 150-300 mg/dl for all ages. Younger people should not exceed 200 mg/dl. In our study, the mean age was 20.7 years and accordingly, the upper limit of normal should be 189 mg/dl (Keys) to 240 mg/dl (Pezhick) if 189 mg/dl is used as the upper limit of normal, all mean cholesterol levels during noise exposure are above normal. Even with the higher limits, all values are statistically significantly elevated from the mean, pre-exposure level, and they begin to decrease after the noise exposure ceases. In this case, the subjects acted as their own controls since all other factors including confinement, diet, and lack of exercise, permitted for 10 days after the noise was stopped. Noise exposure was the only variable that changed.

These findings support the concept that the physiological effects of noise are more serious than previously supposed, and more studies of the effects of noise exposure are indicated.

REFERENCES


DISCUSSION

Q. (Johnson) Do you feel that noise that is so low in level that it will not damage the auditory system will cause any non-auditory effects?

A. (Central) Perhaps that is a loaded question because, as you know, there is a great deal of work going on to determine a safe level of noise that will not damage the auditory system. Some people think it is 90 dBA, others think it is as low as 20 dBA. If one accepts this later statement of 70 dBA being potentially hazardous, then I think that noise under 70 dBA will not cause any physiological effects. The level of noise at which physiological effects begin, and the seriousness of these effects, is not well-enough measured yet to answer your question. My personal opinion is that somewhere between 75 and 80 dBA, both for hearing and for physiological effects, will turn out to be the critical level. I would like to have Dr. Jensen comment on this question also.

A. (Jensen) One observes vegetative reactions at very low levels of noise. It is only a question what method one uses. For example, electrodermal response, or other sensitive physiological or psychophysiological methods, will show that there are quantitative influences at low noise levels. The question is what is the physiological relevance of these changes? I think the question cannot be answered until now as to where the point is that the normal physiological response is accumulated into a pathological one. This is the question one needs to answer. At the present one has no exact point to state where this begins. It is an increasing continuous augmentation of these reactions.
SECTION 25
### SUMMARY FORM FOR
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

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<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tr>
<td>Ronald R. Chessar, Ronald S. Caldwell, and Michael J. Harvey</td>
<td>Ecological Research Center, Dept. of Biology, Memphis State University, Memphis, Tenn.</td>
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### Investigator's Phone No. | Sponsoring Organization |
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### Citation |

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### Purpose for Study |
- To see if adrenal size differences exist between noise-exposed and non-noise-exposed populations of wild mice; to test if these differences are due to noise.

### Description of test groups (subjects, etc.) |
- Feral *Mus musculus*—wild house mouse. Part I—experimental group—field near aircraft—10 adult males, 32 adult females; control group—field near rural field—13 adult males, 13 adult females.
- Part II—experimental group—10 adult males, 4 adult females; control group—9 adult males, 5 adult females.

### Control of other stressors |
- Stress of snap-trapping (which may not kill instantly) was not considered.

### Noise Stimulus |
- Source: I. aircraft; II. recorded aircraft
- Spectral characteristics: not given
- Noise level: I—100 dB avg. aircraft noise II—105 dB
- Length of exposure: 1 min. every 6 min. (II)

### Number of trials |
- Part II—lab simulation of 105 dB—1 minute every 6 minutes for 2 weeks.

### Author's conclusions |
Noise was the dominant stressor that contributed to the greater adrenal size in populations of wild mice near Memphis International Airport than in rural mouse populations.

### Evaluation & comments |
Summary form only
EFFECTS OF NOISE ON FERAL POPULATIONS OF MUS MUSCULUS

RONALD K. CHESSER, RONALD S. CALDWELL, AND MICHAEL J. HARVEY
Ecological Research Center, Department of Biology, Memphis State University

House mice, *Mus musculus*, were snap-trapped from two similar fields near Memphis International Airport, Shelby County, Tennessee, the only apparent difference being presence or absence of low-flying aircraft. Airport field noise levels ranged from 80 to 120 dBA while rural field levels ranged from 80 to 85 dBA. Mice from the airport field had significantly larger adrenal glands than those of the rural field. To determine if noise was the causative stressful factor, mice collected from the rural field were subjected to recorded jet noises at 105 dBA in the laboratory for 2 wk. The experimental group had significantly larger adrenals than those of a control group.

INTRODUCTION

Investigations into the effects of noise on wild populations of animals are few, although laboratory studies are better documented. Fletcher and Harvey (1971) reviewed the literature concerning the effects of noise on wildlife and other animals. Greaves and Rowe (1969) subjected feral *Mus musculus* to noise levels between 85 and 120 dBA in the laboratory. High noise levels deterred mice from entering rooms from which noise was emitted, especially when outside food availability was low. Marsh, Jackson, and Stell (1962) and Sprock, Howard, and Jacob (1967) found that noise had no dispersal effect on feral rodents.

Stress does not lend itself to quantification. However, one measurement of stress is adrenal hypertrophy which adapts the animal to stressful situations (Sawin 1939). Adrenal weights were utilized by many previous experimenters (Christian 1955; Davis and Christian 1957; King 1957; Bronson and Elfr genesis 1963; Southwick 1964) as an indication of stress. Anthony and Ackerman (1955) subjected laboratory strains of *M. musculus* to 110 dBA noise levels and found slight but insignificant adrenal hypertrophy. However, no previous worker has studied adrenal weights under field situations involving stress. We wished to determine if adrenal weight differences existed in two field populations of *M. musculus*, one subjected to a possible stressful factor of noise. If adrenal differences did exist, we wished to determine if the differences were caused by noise.

MATERIAL AND METHODS

Thirty-two adult male and 10 adult female *Mus musculus* were collected from a field approximately 90 m from the end of a runway at Memphis International Airport, Shelby County, Tennessee. Thirteen adult males and 13 adult females were collected from a rural field 2.0 km from the airport field. These two fields were chosen due to similarity in habitat, close proximity, and dissimilarity in noise levels.

Differential noise levels of the fields were measured with a sound survey meter. Background noise levels of the fields were approximately equal, ranging from 80 to 85 dBA. Noise levels of incoming and outgoing aircraft at the
airport field averaged 110 dBA with the highest reading at 120 dBA. Aircraft noises at the rural field were negligible over background. Mice for the field study were snap-trapped on November 26, 1973. Mice were sexed, weighed, and their adrenal glands removed and weighed in the laboratory. Total body weights were measured on a balance beam to the nearest 0.01 g. Adrenal weights were measured on a semiquantitative balance to the nearest 0.1 mg.

To determine if noise was causative, 28 additional mice were live-trapped January 10, 1974, from the rural field and taken to the laboratory. These mice were distributed into two cages measuring 146 X 98 X 60 cm. The experimental group contained 10 males and four females. The control group contained nine males and five females. After an acclimation period of 10 days, experimental mice were subjected to 1 min of 105 dBA recorded jet aircraft noise every 6 min. Control mice were not subjected to jet noise. After 2 wk, adrenals were removed and weighed. Comparisons were made between adult male and female mice. Mice were considered adults if their body weights were 9.5 g or larger (Baker 1946).

Comparisons were made of gross adrenal weights, and differences were tested using Student's t-test. No comparison of adrenal weights to body weights was necessary due to insignificant differences in body weights of the different groups ($P < .05$).

**RESULTS AND DISCUSSION**

Adrenal weights of both male and female mice from the airport field were significantly greater than those from the rural field. In the laboratory study, experimental males and females had significantly greater adrenal weights than control mice (Table 1). From these data we conclude that noise was the dominant stressful factor contributing to the adrenal differences between these two rural populations of *Mus musculus*.

In the field, other factors such as population density, air pollution by jet engines, or sight of aircraft may contribute to adrenal hypertrophy. Of the aforementioned, population density would probably exert greater stress. However, mark-recapture analysis indicated population density of the airport field was not greater than that of the rural field.

**TABLE 1**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Adrenal weight (mg)</th>
<th>Adrenal weight (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>X ($\pm s$)</td>
<td>Y ($\pm s$)</td>
</tr>
<tr>
<td>-----</td>
<td>---------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td></td>
<td>Airport Field (110 dBA)</td>
<td>Rural Field (58 dBA)</td>
</tr>
<tr>
<td>M</td>
<td>12</td>
<td>3.5 ± 0.10</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>4.2 ± 0.17</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>3.3 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>4.3 ± 0.43</td>
</tr>
</tbody>
</table>

Note: Adrenals of all mice were subjected to high noise levels were significantly different from those of the controls at least at the 95% confidence level.

**REFERENCES**

LITERATURE CITED


SECTION 26
# SUMMARY FORM FOR STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

## Principal Investigator(s)
R. D. Fell, C. J. Ellis, D. R. Griffith

## Institution and address where research was performed
Department of Zoology
Iowa State University
 Ames, Iowa 50011

## Investigator's Phone No.

## Sponsor's Organization

## Citation

## # of Ref.'s
2

## # of Fig.'s
2

## Language
English

### Purpose for study
Since other stressors suppress thyroid activity through release of ACTH, the effects of noise-stress on thyroid responses (iodine uptake and body wt.) were studied for comparison purposes.

### Type & duration of experiment
- Laboratory Experiment - 12 weeks
- Soundproof room

### Description of test groups (subject & age, etc.)
100-150 day-old male and female white rats (Sprague-Dawley strain)
- Test Group 1: exposed to noise for 12 weeks
- Test Group 2: exposed to noise for 2 weeks
- Control Group: no noise 12 weeks

### Control of other stressors
- Laboratory conditions; temperature 80°F; sound level - 65 dB background

### Noise Stimulus
- Source: single tone from audio-generator
- Spectral characteristics: 1000 Hz, monotonous
- Noise level: 95 dB
- Length of exposure: 15 minutes
- # of trials: every 15 min. daily for 8 hrs.
- Each trial: 15 min. noise and 15 min. quiet for 8 hrs. daily

### Statistical Methods
- Standard error

### CVS Response Measured
- None

### Nonauditory effects
- Thyroid histology - no significant changes in follicle cell size.
- Iodine uptake - reduced due to noise
- Weight gain - noise induced suppression of wt. gain compared to the controls

### Author's conclusions
Noise stress significantly suppressed cumulative weight gains in both test groups. Weight was suppressed earlier (in the first 2 weeks) in female rats than in male rats (in the 6th week). The weight suppression was correlated with suppression of iodine uptake by the thyroid during the same time periods.

### Evaluation & comments
The use of rats in noise studies is considered suspect by many researchers. The number of animals in each group was not specified.
Thyroid Responses to Acoustic Stimulation

RONALD D. FELL, CHARLES J. ELIAS, AND DAVID R. GRIFFITH

Department of Zoology, Iowa State University, Ames, Iowa 50011

Received October 28, 1975

Male and female rats were subjected to monotonic, 1000 Hz, 95 dB(A), noise stress presented in 15 minute intervals 8 hours per day for 12 weeks. Body weights and thyroid I-131 uptake values were recorded. Relative body weight-change rates were significantly reduced. Female rate of weight-change decreased during the first 2 weeks, and remained lower than controls. Male rate of weight-change decreased during Week 6 and remained low throughout the last 6 weeks. Thyroid I-131 uptake values were low for both sexes and a positive correlation between the time of decreased iodine uptake and suppressed weight-change rates was noted.

INTRODUCTION

Undesirable sound is one of the more recently recognized forms of stress, particularly in the form of daily exposure to environmental noise (Caudron, 1972; Jansen, 1969; Kerbiss, 1972; U. S. Environmental Protection Agency, 1971). This stress is potentially of great detriment to biological systems (Welch and Welch, 1970).

The most common effect cited in past noise-stress studies is adrenocortical activation via the hypothalamic–pituitary axis (Anthony, 1973). Frequencies of 125, 1000, 3000, and 10,000 Hz at levels between 65 and 93 dB caused substantial elevations (twice normal levels) in human free plasma 17-hydroxycorticoids (Arguelles et al., 1962). Urinary 17-ketogenic steroid excretion was also markedly elevated. Apparently, adrenocortical function in humans is remarkably sensitive to auditory stimulation probably through the effect of ACTH released by the stress of noise perception.

Another investigation (Henchin and Knigge, 1963) showed a triphasic adrenocortical response in female rats exposed to sound-stress (110 dB, 220 Hz). Initial high adrenal corticosterone secretion was followed by a period of decreased secretion, which was followed by a return to high output levels. This study correlated with another (Geber et al. 1966) which reported a biphasic adrenal ascorbic acid depletion due to noise-stress (73–93 dB, 20–25 kHz). An initial decrease in ascorbic acid and adrenal weight followed by a rise in ascorbic acid and adrenal weight above control values was observed. These remained high for the experimental period (21 days).

Activation of adrenocortical secretions because of noise-stress correlated with other forms of stress which cause similar results. It has long been known various stressors (legal fracture, formalin injection, shock, surgery, ether, etc.) cause increased adrenocortical hormone secretion concomitant with increased release of ACTH.

Increased secretions of adrenocortical hormones and ACTH have been related to decreased thyroid function due to suppressed TSH release from the

200
adenohypophysis (Brown-Grant et al., 1954; Harris, 1955; Knigge, 1960; Nicoloff et al., 1970; Wilber and Utiger, 1969). Although the mechanism of this action is obscure, evidence has accumulated favoring a common precursor for ACTH and TSH (DeGhoom et al., 1966; Guillemin, 1968; Reifuna et al., 1968).

Due to stress release of ACTH and ACH, and their suppressing effects on thyroid activity, examination of the thyroid response to moderate noise-stress seemed appropriate.

METHODS
Throughout this investigation, 100-150 day old albino rats raised in our laboratory (Sprague-Dawley origin) were used. They were maintained on Wayne lab chow and water ad libitum. Male and female animals were divided into one control and two experimental groups.

Control rats were kept in a moderately soundproof room where ambient conditions of temperature (80°F), light cycle (12 hours), and sound level, 68 ± 2 db(A), existed. Experimental rats were subjected to sound-stress in an environmental chamber (7 x 12 x 10 ft) where ambient conditions identical to the control room existed.

A single tone (1000 Hz), recorded from a Heathkit IG72 audio-generator, was played to experimental animals in 15 minute periods followed by 15 minutes of ambient sound conditions. This routine was continued during the light phase 8 hours per day for 12 weeks. Group I experimental animals remained in the chamber 12 weeks. Group II experimental animals were placed in the chamber at the start of the 10th week, therefore only receiving sound-stress for 2 weeks.

During the experimental period all animals were observed for signs of otitis media, and at time of sacrifice, 25% of the animals were randomly inspected for visual indications of middle ear infections. Based on these inspections, animals were found to be free of gross hearing abnormalities.

Throughout this experiment body weights were measured weekly. At termination of the 12th week of sound-stress all rats were lightly anesthetized with ether and injected with five µCi of I-131 per animal. All animals remained in the radioisotope control room 24 hours. At the end of this period they were again lightly anesthetized and external thyroid counting, utilizing a Nuclear Chicago 1620A countmeter meter, was carried out. A percentage of iodine uptake was calculated and significance testing carried out by the Iowa State University Statistical Laboratory. Animals were sacrificed after external thyroid counting. Thyroid glands were excised and fixed in 10% buffered formalin and embedded in paraffin for later histological examination.

RESULTS
Mean cumulative weight gain of all groups appear in Table 1. Control animals exhibited increases in weight throughout the experiment. Control males averaged 242 g at Day 1, and gained weight to an average of 427 g at sacrifice. Control females averaged 234 g at Day 1 and after 12 weeks averaged 424 g. The rate of gain for females slowed during the last several weeks.

Group II female rats averaged 259 g at the onset of sound-stress and after 12 weeks of it their average weight had increased only to 264 g. At no time during the
TABLE I
MEAN CUMULATIVE WEIGHT GAINS (GRAINS) ± STANDARD ERROR

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
<th>Week 4</th>
<th>Week 5</th>
<th>Week 6</th>
<th>Week 7</th>
<th>Week 8</th>
<th>Week 9</th>
<th>Week 10</th>
<th>Week 11</th>
<th>Week 12</th>
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<td>CFT</td>
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<td>16.9</td>
<td>27.6</td>
<td>39.8</td>
<td>52.7</td>
<td>57.6</td>
<td>66.8</td>
<td>78.6</td>
<td>93.6</td>
<td>108.0</td>
<td>116.0</td>
<td>119.0</td>
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<td>CMT</td>
<td>30.7</td>
<td>54.4</td>
<td>67.1</td>
<td>82.3</td>
<td>91.1</td>
<td>111.3</td>
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<td>133.7</td>
<td>151.6</td>
<td>163.0</td>
<td>165.0</td>
<td>168.0</td>
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<tr>
<td>BFT*</td>
<td>6.0</td>
<td>±1.8</td>
<td>±2.1</td>
<td>±2.6</td>
<td>±2.5</td>
<td>±3.7</td>
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<td>±5.9</td>
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<td>1.6</td>
<td>6.2</td>
<td>±2.3</td>
<td>±2.7</td>
<td>±3.0</td>
<td>±3.2</td>
<td>±3.7</td>
<td>±3.8</td>
<td>±3.9</td>
<td>±4.0</td>
<td>±4.4</td>
<td>±4.5</td>
</tr>
<tr>
<td>EMP</td>
<td>6.7</td>
<td>18.8</td>
<td>±3.9</td>
<td>±6.9</td>
<td>±10.3</td>
<td>±11.4</td>
<td>±12.2</td>
<td>±12.8</td>
<td>±15.8</td>
<td>±13.3</td>
<td>±13.5</td>
<td>±13.8</td>
</tr>
</tbody>
</table>

* Relative to starting weight.
* Control females (ambient sound).
* Control males (ambient sound).
* Group I—Experimental females (sound stressed 12 weeks).
* Group I—Experimental males (sound stressed 12 weeks).
* Group II—Experimental females (sound stressed 2 weeks).
* Group II—Experimental males (sound stressed 2 weeks).

12 week period were significant differences found between any of the weekly weighings. However, when experimental females in group I were compared to control females, highly significant differences (P < 0.001) existed between all 12 week cumulative weight gain average values. The body weight gains of experimental females were significantly suppressed throughout the experiments, in fact, during Week I they lost weight.

Group I male rats were placed in the experimental chamber having an average weight of 316 g and at termination of 12 weeks of sound-stress these animals averaged 425 g. Although these animals gained weight, as did their control counterparts, significant differences (P < 0.05) showed at Week 6 and became increasingly significant until time of sacrifice. A suppressed growth rate was displayed for experimental males and experimental females but for the latter the growth suppression commenced immediately after being subjected to sound-stress.

Group II rats were exposed to sound-stress for only 2 weeks. Females began their stress period averaging 236 g and after 2 weeks in the chamber weighed 242 g. Males averaged 320 g when initially placed in the chamber and after 2 weeks of sound-stress weighed 339 g.

When compared to control animal cumulative weight gains, highly significant differences (P < 0.001) were found in both males and females.

Group II females did not lose weight, but group I females showed suppressed growth immediately after sound-stress began. Males in group II did not continue gaining as control animals. Instead their growth was suppressed at once but not to the extent of females in the same group.

Percent first-131 uptake for 24 hours (Table 2) showed a significant sex difference among control rats (P < 0.01) and a similar sex difference among group II rats (P
THYROID RESPONSES

TABLE 2

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean percentage uptake</th>
<th>Standard error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control females</td>
<td>1.53</td>
<td>0.28</td>
</tr>
<tr>
<td>Control males</td>
<td>2.48</td>
<td>0.22</td>
</tr>
<tr>
<td>Group I females</td>
<td>2.32</td>
<td>0.14</td>
</tr>
<tr>
<td>Group I males</td>
<td>1.57</td>
<td>0.11</td>
</tr>
<tr>
<td>Group II females</td>
<td>2.56</td>
<td>0.21</td>
</tr>
<tr>
<td>Group II males</td>
<td>2.49</td>
<td>0.28</td>
</tr>
</tbody>
</table>

* Group I animals were sound-stressed 12 weeks, group II rates exposed only 2 weeks.

< 0.05). No significant sex difference was shown among group I animals.

A significant decrease in iodine uptake resulted between control females and group I females ($P < 0.01$). The difference in this uptake between group II females and control females was also highly significant ($P < 0.001$). However, no significance was recorded between females in groups I and II.

Control males and group I males displayed no difference in I-131 uptake. Yet a highly significant difference existed between control males and group II males ($P < 0.001$). Only a significant difference was noted between group I males and group II males ($P < 0.02$).

Female iodine uptake values were reduced in this study sometime during the first 2 weeks of sound exposure. Their uptake stayed at that level throughout the remaining 10 weeks. On the other hand, male iodine uptake was equal and constant in level the first 2 weeks of the study but decreased sometime between the 2nd and 12th weeks (Table 2).

Thyroid glands were sectioned and analyzed for follicle and follicle cell size. No significant alterations in structure or size were noted.

**DISCUSSION**

This study shows growth rates are altered by sound-stress at the level reported herein and that these alterations are sexually differentiated both in amount and time of response. Suppression of cumulative growth rates in female rats occurred more rapidly than in males and appeared to be more severe.

Depression of body weight has been demonstrated using various types of stress and attributed by some (Sackler et al., 1959; Sackler et al., 1960) to decreased food intake during the stress, as well as other hormonally induced factors. Results presented in this paper reveal a close correlation between decreased relative weight gains and depression of I-131 uptake values for both male and female rats. Females lost weight during the first 2 weeks, and their iodine uptake values were suppressed during this same period. Males began to lose weight significantly during the sixth week of sound-stress. Their iodine uptake values were depressed during the last 10 weeks. This depressed I-131 uptake may have been initiated during the sixth week.

Suppressed body weight gains of stressed animals, correlated chronologically with decreased I-131 uptake values (groups I and II), suggest a physiological difference in the response to sound-stress between male and female rats.
Sound of various levels has been used by other investigators to observe stress reactions (Anthony, 1973; Arguelles, 1962; Geber et al., 1966; Henkin and Knigge, 1963). A stress response to sound elicits higher levels of ACTH and the catabolic glucocorticoids. Adrenal cortical hormones have an inhibitory effect on the thyroid gland or anterior pituitary release of TSH (Brown-Grant et al., 1954; Knigge, 1960; Nicoloff et al., 1970; Wilber and Uliger, 1969). Another suggested possibility is that sound through extrahypothalamic or hypothalamic influences reduced TSH secretion by the anterior pituitary and thus indirectly altered thyroid secretion. This altered thyroid function may in turn decrease the secretion of growth hormone, antagonize the effects of growth hormone, or decreased thyroid function may alter the synergism between thyroid hormone and growth hormones (Daughaday et al., 1975). These endocrine interactions, along with decreased food intake, are possible explanations for the loss of weight gain observed in these rats.

Parameters measured in this investigation (relative weight gain and iodine uptake values) exhibited sex differences. Gonadal hormones may play a role in these differences since gonadotrophin-secreting pituitary basophils increase in number under sound-stress (Suckler et al., 1959; Suckler et al., 1960). Ovaries increased in size and weight, uteri decreased in weight, whereas males exhibited no testicular change in normal spermatogenesis during such stress (Zondek and Tamari, 1964). Along with these anatomical effects, decreased fertility rates have been reported among similarly stressed rats (Zondek and Tamari, 1964). The mechanism of gonadal hormone involvement is still unknown and highly variable with the amount and level of sound-stress used.

Results of this investigation emphasize more research is needed to examine effects of sound-stress on both male and female physiological and endocrinological systems, specifically in regard to thyroid function.

REFERENCES
THYROID RESPONSES


SECTION 27
STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

**Principal Investigators:**
D. Guha, et al.

**Institution and address where research was performed:**
Department of Pharmacology
Howard University College of Medicine
Washington, D.C. 20059

**Citation:**

**Type & duration of experiment:**
Laboratory experiment in sound-proof chamber; 4 hour sessions - 32 days total

**Purpose of study:**
To determine the effects of noise on gastric secretion, and correlate these effects with blood corticosterone levels

**Description of test groups (Subjects, n's, age, etc.):**
250 - 300 g Wistar rats (including unspecified numbers of males and females) with implanted stomach cannulas. The test and control groups contained 6-8 rats each.

**Control of other stressors:**
Implanted cannulas may have added additional stress; experiments began 2 weeks post-operative

**Noise Stimulus:**
Source: through a speaker
Spectral characteristics: 4000 cycles per second
Noise level: 80 dB
Length of exposure: continuous for 1 or 2 hr.
No. of trials:
Phase A - quiet in tests and controls
Phase B - noise for 1 or 2 hr. in tests
Phase C - quiet in tests and controls

**Statistical Methods:**
Mean, standard error, and student's t-test

**CVS Response Measured:**
None

**Nonauditory effects:**
Significant decrease in gastric secretion and increased corticosterone levels during periods of noise exposure, returning to normal in one hour.

**Author's conclusions:**
Continuous exposure to noise stress produced a significant decrease in the volume of gastric secretion and an increase in the plasma corticosterone level. An increase in gastric secretion occurred in the first hour before the noise stimulus (in phase B), an anticipatory mechanism that may be adaptive.

**Evaluation & comments:**
This article supports the idea that noise stress could be a cause of stomach ulcers by decreasing the volume of gastric secretion, which increases stomach acidity. Evidence that noise acted as a stressor is indicated by increased plasma corticosterone levels.
EFFECTS OF SOUND STIMULUS ON GASTRIC SECRETION AND
PLASMA CORTICOSTERONE LEVEL IN RATS
Debjanl Guha, Evan F. Williams, Yuth Nimitkitpaisan,
Sikta Bose, S. M. Dutta and S. N. Pradhan
Department of Pharmacology
Howard University College of Medicine
Washington, D.C. 20060

Abstract
The effects of sound stimulus were studied on the gastric secretion
in rats with chronically implanted cannulas. Attempts were made to correlate
the changes in the secretion with those of the plasma corticosterone level.
Exposure of the animals to sound stimulus (1 hr or 2 hr) produced a marked
decrease in gastric secretion and a concomitant increase in plasma corti-
costerone. It appears that in producing these effects sound stimulus acted
as a stressor. Furthermore, a paradoxical increase in secretion was noted
in the first hour collection prior to the sound stimulus. This initial
increase in secretion may be due to an adaptive compensatory mechanism in
anticipation of the marked inhibition.

Introduction
Continuous exposure to sound stimulus has been shown to produce certain
disease states such as peptic ulcer, hypertension and facial skeletal defects
gastric ulcer has been reported to occur as a result of increased stomach
acidity concomitant with a marked decrease in the volume of gastric secretion
(Frodie et al., 1962). Studies from this laboratory have also demonstrated
that different types of stress including sound stimulus inhibit the growth of transplanted 4M mammary carcinomas and 7,12-dimethylbenz[a]anthracene-induced tumor (Fradhan and Ray, 1974). Rosecrans et al. (1966) observed an increase in both plasma corticosterone and urinary catecholamines on exposure to combined auditory, visual and motion stress.

The present study was therefore undertaken to determine the effects of chronic exposure to auditory stimulus on the volume of gastric secretion in rats. Attempts were also made to correlate the stress-induced changes in gastric secretion with blood corticosterone level.

Methods and Materials

Rats of Wistar strain weighing 250-300g and of either sex were used. The animals were implanted with stainless steel cannula in their stomach according to the method of Pare (1972) for repeated collections of gastric secretion over prolonged periods (Gaba et al., 1974). Experiments were started following a post-operative period of 2 weeks during which rats were also adapted to the experimental situations. The experimental and control groups each consisted of 6-8 rats.

During a daily session lasting for 3 or 4 hours animals were kept inside a sound-proof cabinet fitted with a speaker and gastric secretion was collected every hour. Sessions were carried out 6 days a week at the same hour of the day.

Sound stimulus (80 db, 4000 cps) was presented to the experimental group during the 2nd hour in two experiments (I and II) and during the 2nd and 3rd hours in one experiment (III); these sound exposure periods were preceded and followed by an hour of collection of control secretion.

Gastric secretion was experimental period with presence of sound stimulus period without any hour, and phase C, p: detailed analysis of c periods lasting for 3-4 days under same conditions as exp.

Micro-determination to the method of Tuki experimental and control: gastric secretion of paired comparison beta Student's A test.

A. Gastric secretion
(1) Effect of experiments performed
In the control group, 1.2-1.7 ml and did not differ grossly from the experimental group values during phase A. In p.

Effectively reduced in t.

3 ml to its control value showed a progressive
Gastric secretion was measured during the sessions throughout the whole experimental period which was divided into three phases depending on the presence of sound stimulus in the experimental group: phase A, control period without any sound; phase B, exposure to sound stimulus for 1 or 2 hours; and phase C, post-stimulus control in absence of sound. For detailed analysis of data each of these phases was further subdivided into periods lasting for 1-6 days as shown in Tables 1 and 2. In each experiment, the gastric secretions of a control group were monitored under the same conditions as experimental rats but without exposure to sound.

Hormone determination of the plasma corticosterone was done according to the method of Kisch et al. (1974) at the end of each phase in both experimental and control groups. The mean and standard error of the hourly gastric secretion in different phases were determined in both experiments. Paired comparison between the data from the relevant groups was done by Student's t test.

Results
A. Gastric secretion

1. Effect of one-hour sound exposure. The results of one of the two experiments performed with one-hour sound exposure, are presented in Table 1. In the control group, the hourly secretions in three phases ranged from 1.2-1.7 ml and did not differ significantly from each other. In the experimental group there was also no apparent difference in the hourly secretions during phase A. In phase B, following exposure to sound, the secretion was markedly reduced in the 1st hour. In the 2nd hour, the secretion returned to the control value as in phase A. The 3rd hour secretion in this phase showed a progressive increase over that in phase A. In phase C, the hourly
TABLE 2. Effect of 2-hour sound exposure on gastric secretion* in rats.

<table>
<thead>
<tr>
<th>Phase</th>
<th>Experimental group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.5 ± 0.1</td>
<td>1.6 ± 0.6</td>
</tr>
<tr>
<td>2</td>
<td>1.4 ± 0.01</td>
<td>1.5 ± 0.1</td>
</tr>
<tr>
<td>3</td>
<td>1.7 ± 0.02</td>
<td>1.5 ± 0.03</td>
</tr>
<tr>
<td>4</td>
<td>1.9 ± 0.02</td>
<td>1.7 ± 0.05</td>
</tr>
<tr>
<td>5</td>
<td>2.0 ± 0.02</td>
<td>1.9 ± 0.02</td>
</tr>
</tbody>
</table>

*Significantly (p < 0.01, except for 1st hour secretion during the 1st 6 days of sound exposure).

In the 4th hour (after the removal of sound), the values of secretion returned to 1st hour level, except for a slight increase in the 4th hour of phase 2. In the 4th hour (after the removal of sound), the values of secretion returned to 1st hour level, except for a slight increase in the 4th hour of phase 2.
TABLE 2. Effect of 2-hour sound exposure on gastric secretion\(^a\) in rats.

<table>
<thead>
<tr>
<th>Phases and Days</th>
<th>Experimental group(^b)</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1st hr.</td>
<td>2nd hr.</td>
</tr>
<tr>
<td>A 1-5</td>
<td>1.9 ± 0.1</td>
<td>1.9 ± 0.2</td>
</tr>
<tr>
<td>6-10</td>
<td>1.2 ± 0.01</td>
<td>1.0 ± 0.1</td>
</tr>
<tr>
<td>B 1-5</td>
<td>1.6 ± 0.2</td>
<td>1.3 ± 0.2(^c)</td>
</tr>
<tr>
<td>6-10</td>
<td>1.7 ± 0.02</td>
<td>0.6 ± 0.2</td>
</tr>
<tr>
<td>11-15</td>
<td>1.6 ± 0.2</td>
<td>0.9 ± 0.2</td>
</tr>
<tr>
<td>C 1-6</td>
<td>1.5 ± 0.1</td>
<td>1.5 ± 0.2</td>
</tr>
</tbody>
</table>

\(^a\) ml/hour (mean ± S.E.).

\(^b\) Rats of this group were exposed to sound during the 2nd and 3rd hours of phase B.

\(^c\) Significantly (P < .001) less than the corresponding 1st hour secretion.
controls in phase A. In phase C, all hourly secrections returned very close
to their control values as in phase A.

B. Plasma corticosterone level.

In the experimental groups of rats plasma corticosterone levels increased
significantly after sound exposure for 1 hour (in experiment I) or 2 hours,
( in experiment III) in phase B, while there was no significant change in its
levels in the control groups (Table 3). During the later phases due to
technical difficulties the plasma corticosterone levels could be correctly
measured only from a limited number of rats. These data are scarce and variable
and not presented here.

<table>
<thead>
<tr>
<th>Experiment No. and phases</th>
<th>Experimental group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experiment I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase A</td>
<td>8,5 ± 0,5 (6)</td>
<td>8,8 ± 0,3 (4)</td>
</tr>
<tr>
<td>Phase B</td>
<td>14,6 ± 0,5 (7)</td>
<td>9,6 ± 0,4 (4)</td>
</tr>
<tr>
<td>Experiment III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phase A</td>
<td>5,7 ± 0,5 (7)</td>
<td>5,8 ± 0,3 (4)</td>
</tr>
<tr>
<td>Phase B</td>
<td>7,1 ± 0,4 (5)</td>
<td>5,2 ± 0,5 (4)</td>
</tr>
</tbody>
</table>

\(^{a}g/20\mu l; mean ± S.E.

\(^{b}\)Rats were exposed to sound for 1 or 2 hours
during phase II of Experiments I and III
respectively.

\(^{c}p < .001; \ ^{d}p < .05.\)
Discussion

Repeated exposure of rats to auditory stress produced significant decrease in the volume of gastric secretion concomitant with a marked increase in the plasma corticosterone level. These findings are in close agreement with the results of other investigators who showed that environmental 'stress' of any kind not only decreased the gastric secretion, but also increased the serum cortisone level (Brodie et al., 1961; Brodie and Hoek, 1971; Dai and Ogle, 1973; Snookler and Buckley, 1970; Mikhail, 1971; Friedman and Ader, 1965;ears, 1964). If the stress is continued for a prolonged period, its inhibitory effect on gastric secretion gradually becomes less. This decrease of sound-induced inhibition of secretion may have resulted from the development of tolerance to sound or exhaustion of available elements of gastric secretion, as reported by Foris (1967) in relation to drug-induced changes.

Many hypotheses have been put forward to explain the mechanism for the stress-induced decrease in gastric secretion (Dai and Ogle, 1974; Takagi and Okabe, 1970). This decrease induced by sound stress may be due to transient ischemic change in the gastric mucosa (Goldman and Rosoff, 1968) and to increased gastric motility produced during stress (Watanabe, 1966).

Sound-induced changes in blood steroid levels observed in this study closely agree with those of Snookler and Buckley (1970). They reported an increase in blood steroid following auditory stress in rats concomitant with hyperfunction in all 3 zones of the adrenal cortex. They also suggested that during the sound exposure central noradrenergic neuronal centers could have been stimulated resulting in liberation of ACTH from the anterior pituitary leading to elevation of adrenal steroid secretion.
During the first hour of phase B of these experiments the gastric secretion showed a progressive increase. This paradoxical increase was similar to that observed in relation to inhibition of gastric secretion by Guha et al. (1974) following epinephrine treatment and by Kahl (1952) during chronic fear. This increase may be attributed to an adaptive compensatory mechanism in anticipation of its marked inhibition induced by the stress (Guha et al., 1974).

Acknowledgement

The authors are thankful to Dr. Rajendra Kumar for his assistance in estimating the plasma corticosterone levels in the later part of this work.

References


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**Summary Form for Studies on the Effects of Noise on the Cardiovascular System (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>J. D. Hanson, N. E. Larson, and T. Snowdon</td>
<td>Department of Psychology, University of Wisconsin, Madison, Wisconsin 53706</td>
</tr>
</tbody>
</table>

<table>
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<th>Investigator's Phone No.</th>
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<td></td>
<td>National Science Foundation</td>
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<td>NSF Grant GY-1159</td>
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| Citation | Hanson, J. D., et al. The effects of control over high intensity noise on plasma cortisol levels in Rhesus monkeys. Behavioral Biology 16:333-340, 1976. |

<table>
<thead>
<tr>
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<th># of Fig's</th>
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<th>English</th>
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</table>

<table>
<thead>
<tr>
<th>Type &amp; duration of experiment</th>
<th>Purpose for study</th>
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</thead>
<tbody>
<tr>
<td>Type: Laboratory experiment in a soundproof chamber</td>
<td>To study the effects of control over a stressful stimulus (noise) on plasma cortisol levels</td>
</tr>
<tr>
<td>Duration: 11 days - monkeys trained to control noise. 28 days - experiment</td>
<td></td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Description of test groups (subjects, #, sex, etc.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 Rhesus monkeys: 12 one-year olds (6 males, 6 females) 12 3-year olds (6 males, 6 females)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Three groups:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. no noise 2. control over noise 3. loss of control over noise (same animals as group 2)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Control of other stressors: Laboratory conditions; some stress may have been associated with venipuncture</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Noise Stimulus</th>
<th>Statistical Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source: (power tools, heavy machinery, drills)</td>
<td>2x2x3 factorial design; Fisher LSD test; analyses of variance</td>
</tr>
<tr>
<td><em>Ampex Model AG 600 tape recorder</em> spectral characteristics: reported in previous paper noise level: 100 db</td>
<td></td>
</tr>
</tbody>
</table>

| Length of exposure: 4 23-min. noise periods with 2 min quiet periods in between 20 trials | CVS Response Measured |
|==================================================================|-------------------|
| 2 | none |

<table>
<thead>
<tr>
<th>Nonauditory effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Increased plasma cortisol levels in animals with no control over the noise</td>
</tr>
<tr>
<td>2) Levels of plasma cortisol in animals with control over noise were similar to that of controls with no noise</td>
</tr>
<tr>
<td>3) Aggressive behavior increased in animals with loss of control over noise</td>
</tr>
<tr>
<td>4) Anti-social behavior (decreased social contact) was greater in animals with no control over noise</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Author's conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monkeys who had control over high intensity noise had blood cortisol levels similar to that of the controls (no noise). Loss of control over noise increased blood cortisol levels to that of the monkeys who never had control over noise. Cortisol levels may be a reliable measurement of the short term effects of stress in humans.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Evaluation &amp; comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>This research is mainly concerned with the adverse effects of noise stress on primate behavior and is a fairly well controlled study.</td>
</tr>
</tbody>
</table>
The Effects of Control over High Intensity Noise on Plasma Cortisol Levels in Rhesus Monkeys

JOHN D. HANSON, MARK E. LARSON, and CHARLES T. SNOWDON

Department of Psychology, University of Wisconsin, Madison, Wisconsin 53706

The effects of control over exposure to high intensity noise on plasma cortisol levels and social behaviors were examined in rhesus monkeys. There were four conditions: control over noise, loss of control over noise, no control over noise, and no noise. Plasma cortisol data indicated that animals with control over high intensity noise stimulation did not differ from animals exposed to no noise at all. Plasma cortisol levels were significantly elevated in animals with no control over high intensity noise and in animals experiencing a loss of control over noise. Animals which experienced loss of control over noise showed increased aggressive behavior while animals with no control over noise showed significantly less social contact than other animals.

In recent years considerable attention has been given to research involving the effects of control over aversive stimuli both with human and nonhuman subjects. Research with humans has used electric shock or high intensity noise as the aversive stimulus and CSR, self-report, and performance tasks as measures of stress. While the results with CSR measures have been contradictory, task performance seems generally to have been less impaired when the subjects perceived that they had control over the stressful stimulus than when they had no control over the stressful stimulus (Bandler, Madras, and Bem, 1968; Corah and Boffa, 1970; Champion, 1950; Geer, Davidson, and Gatchel, 1970; Class, Reim, and Singer, 1971; Staub, Tursky, and Schwartz,

1This is publication 15-030 of the Wisconsin Regional Primate Research Center. This work was supported by NSF Grant 07-1519 (Student Originated Studies). We are grateful to Dr. R. W. Guy, Director of the Wisconsin Primate Center, for making available to us the animals and facilities of the Center. We wish to acknowledge the contributions of the other members of the project: John Cotton, Melanie Ignjatovic, Charles Juno, Miso Kuosia, Michele Peters, Stanley Urban, Lawrence Wasserman, and Hannah Wu. We are also grateful for the advice and assistance of R. E. Bowman, W. D. Houser, F. M. Neals, and W. W. Hugdahl.

2Address reprint requests to Charles T. Snowdon, Psychology Department, University of Wisconsin, Madison, 53706.

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1971. Lefcourt (1973) reviewing the results of several studies has argued that the perception of control over an aversive stimulus is a salient feature in the reduction of stress reactions to the aversive stimulus.

Research conducted using non-human subjects concerning the effects of control over stress-causing stimuli has been limited and contradictory. Brady (1956) using rhesus monkeys and electric shocks, reported development of gastro-intestinal ulcers among animals with control over shock presentation whereas no such ulceration developed among animals with no control over shocks. Weis (1968) subjected rats to severe electric shocks and reported finding ulcers in the rats with no control over the administration of shocks and no ulcers in rats with control, a direct contradiction of Brady.

The present study used plasma cortisol levels as a physiological measure rather than ulceration or GSR skin conductance measures. While gastro-intestinal ulceration is recognized as a long term stress index our concern was with the short term effects of exposure to stress. GSR measures have produced contradictory results whereas plasma cortisol levels provide a high degree of reliability and specificity (Bowman and DeLuna, 1969). This study used continuous high intensity noise as a stressor since it has been shown previously to produce short term increases in cortisol levels in rats (Henkin and Knigge, 1963), in monkeys (Nealis and Bowman, 1972) and in humans (Arguelles, Iheas, Ottone, and Chekherdenian, 1962). Social behaviors were measured immediately following noise exposure. The major concern of the present study is the effect of control over the aversive noise stimulus and the effect of no such control on cortisol levels and on measures of social behaviors.

METHOD

Subjects. Twenty-four rhesus monkeys (Macaca mulatta), 12 one-year olds (six males and six females) and 12 three year olds (six males and six females) were used in this experiment. All monkeys were laboratory born animals raised in small social groups. The monkeys had not previously been exposed to high intensity noise. Feeding, handling, and maintenance schedules were held constant throughout the study.

Apparatus and Materials. The noise stress was modeled after that used by Nealis and Bowman (1972). One hour of continuous noise (power tools, heavy machinery, pneumatic drills, snowmobiles, etc.) at 100 db (re 0.0002 dynes/cm²) was administered in a soundproof chamber (10 X 10 X 8 ft. l.d.). Nealis and Bowman (1972) reported that this continuous noise was the most effective form of high intensity noise and that it produced its maximum effect on cortisol levels after one hour of stimulation. An Ampex Model AG 600 tape recorder was used to record and playback the
auditory stimuli. The noise was amplified by an Altec Model 1594A amplifier and an Electrovoice Sentry IV horn loud speaker system (Frequency response 50-20,000 Hz). Behavioral observations were made in a playroom described by Harlow, Rowland, and Griffin (1964).

*Design.* The experiment employed a $2 \times 2 \times 3$ factorial design. There were two age groups, one and three year olds, and two experimental parts. Each of the experimental parts had three groups. In the first part these groups included control over noise, no control over noise, and a no noise group. In the second part, the monkeys from the control over noise condition lost their control over noise stimulation. The no control over noise animals, and the no noise animals were treated as in the first part. Each monkey was tested twice in each part of the experiment.

*Procedure.* Monkeys in the control over noise group were trained to terminate the noise by depressing a retractable lever. Each animal in the no control over noise group was paired with one in the control over noise group and was exposed to equal durations of noise with the lever made inoperable. At the end of 11 days of training all monkeys in the control over noise group performed perfectly, i.e., upon presentation of the lever, during noise, the subjects depressed the lever within 1-3 sec. No noise monkeys were familiarized with the sound room environment but were never exposed to noise. During this training period, all animals were familiarized with the Playroom.

The experimental conditions were administered to all groups over a period of twenty-eight days. Treatments of specific individual subjects were separated by a minimum of seven days to insure alleviation of any temporary noise-induced stress. The experimental condition consisted of four 13-min periods of noise separated by 2 min intervals of silence. In the control over noise group, the lever was presented at the end of the 13 min noise interval. These trained monkeys always pressed the lever within a few seconds and the noise stopped.

In the no control over noise group, noise was again presented in the four 13 min periods followed by 2 min of silence, however, the retractable lever was not presented. In the no-noise group, animals were exposed to neither the noise nor the retractable lever. In the second part of the experiment the monkeys previously in the control over noise group were now presented with the retractable lever at intermittent times, but pressing the lever did not terminate the noise stimulus. Each animal in this group received two such extinction trials.

Immediately following the experimental treatment, cannulation of the saphenous vein was performed and 1 ml of whole blood was obtained. Centrifugation followed and the resulting plasma was analyzed by the protein-binding radioimmunoassay for cortisol defined by Bowman and DeLuna (1960). At least six replicate cortisol determinations were made on each sample. The cortisol value for each sample was the mean of the first
three determinations that were within 5μg/100ml of each other. Basal non-stress cortisol levels were established on blood drawn 24 hr prior to each noise stress test.

The behavioral activities were scored 1/2 hr after blood drawing in the playroom for 30 min using a modified frequency scoring technique. A modification of the Hanlen-Seay Behavior Definitions (Suomi, Harlow, and Kimball, 1971) with 24 categories was used. Behaviors showing extremely low frequency were dropped from analysis leaving a total of 13 behavior categories. Two observers scored the test animal simultaneously. Three animals of comparable age, weight, and dominance were placed in the playroom with the test animal. The same four animals were always tested in the playroom together.

Prior to the start of the experiment, pre-tests for social behavior measures were conducted in order to familiarize the monkeys with the playroom. Inter-observer reliability was maintained throughout the experiment above the 0.95 level (Pearson product-moment correlation).

Data Analysis. Analysis of variance were employed for evaluating the data of the present study. The Fisher LSD test was used to determine significant differences between means.

RESULTS

Biochemical Data. An analysis of variance of the basal cortisol levels over groups and parts of the experiment showed no significant effects. Since basal levels were equivalent, all subsequent cortisol analyses were based on difference scores. Plasma cortisol difference scores (cortisol levels following noise exposure minus the cortisol levels found 24 hr earlier) for all animals were subjected to an analysis of variance. The Treatment X Parts interaction was significant ($F(2,18) = 7.86, P < .025$) (Fig. 1). The plasma cortisol difference scores of monkeys which had control over high intensity noise did not differ from those of animals which received no noise stimulation at all ($P > .10$). However, the monkeys which received identical amounts of high intensity noise but which had no control over the noise showed significant elevations in plasma cortisol relative to both the no-noise group ($P < .05$) and the control over noise group ($P < .05$). The animals in the loss of control over noise condition showed an elevation of cortisol to levels comparable to animals which had no control over noise ($P < .05$). These levels were significantly greater than the levels found in the same animals when they had control over the noise stimulus ($P < .05$) and significantly greater than animals in the no noise condition ($P < .05$).

Social Behavior Data. Analyses of variance of the measures of social behavior yielded only two significant effects involving control over noise. The
EFFECTS OF CONTROL OVER HIGH INTENSITY NOISE

Fig. 1. Changes in cortisol levels following exposure to high intensity noise. (n = no noise; p = control over noise; p = loss of control over noise; p = no control over noise.)

TABLE I
Frequency of External Aggression Per Session in Three Year Old Monkeys

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mean Frequency</th>
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<tbody>
<tr>
<td>No noise</td>
<td>1.19</td>
</tr>
<tr>
<td>Control over noise</td>
<td>.88</td>
</tr>
<tr>
<td>No control over noise</td>
<td>.63</td>
</tr>
<tr>
<td>Loss of control over noise</td>
<td>2.50#</td>
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*Significantly different from other conditions, P's < .05.

Measure of External Aggression had a significant Treatment x Age x Parts interaction (F(2,18) = 7.50, P < .01). Three year old monkeys in the loss of control over noise condition exhibited higher levels of aggression than animals in all other conditions. Means are presented in Table 1. There was a significant Treatment main effect for Social Contact (F(1,18) = 9.51, P < .01). The animals with no control over noise showed significantly less social contact than animals in the other groups. Means are presented in Table 2. This is
similar to the findings reported by Neils and Bowman (1972) whose noise stressed animals (without control over noise) showed a reduction in social interactions.

DISCUSSION

The control over aversive high intensity noise produced significant reduction of the physiological response to stress as measured by plasma cortisol levels. Changes in cortisol levels relative to non-stress periods were similar in animals with control over the high intensity noise and those who were never exposed to noise at all, while elevated cortisol levels were found in animals exposed to identical amounts of high intensity noise, but who had no control over the presentation of noise. When the animals which had control over the noise stimulus had that control removed, there was an increase in their cortisol difference scores. The levels were significantly higher than they had been when the same animals had control over the noise and were similar to those of noise exposed animals which had never perceived control over the noise. Thus, control over an aversive stimulus markedly reduced the cortisol response that normally accompanies the aversive stimulus.

This increased cortisol response from control over noise to loss of control over noise could not have been due to the passage of time or increased exposure to the noise stimulus, since both the noise animals and the animals with no control over noise exposure showed no cortisol changes over the same period. Variance due to diurnal changes in cortisol levels was controlled in this study since cortisol difference scores were used with the sample taken directly following noise exposure being compared with one taken exactly 24 hr previously when no noise exposure had occurred. Seasonal variations in cortisol levels are likely to have been of little consequence since the entire study was carried out in 28 days during the summer. The cortisol difference levels obtained in the present study from animals in the no noise and the no control over noise condition are similar to those found for
EFFECTS OF CONTROL OVER HIGH INTENSITY NOISE

Comparative treatments of monkeys by Nealis and Bowman (1972), while the
difference level of the loss of control over noise animals is close to the
maximal cortisol response to ACTH injections reported by Meyer and
Bowman (1972). In light of this close correspondence of data from noise
stressed animals with other data, the finding of a low cortisol response in
animals with control over noise is the more striking. Clearly, control over an
aversive noise stimulus can greatly attenuate the cortisol response to the
aversive stimulus.

Some of the behavioral measures further substantiate these findings.
Among three year old animals the loss of control over noise manipulation
produced levels of aggression that were significantly greater than the levels
found in any other condition. The animals in the no control over noise group
showed a significantly lower level of social contact than did animals in other
treatment groups. This finding is similar to the finding of Nealis and Bowman
(1972) that animals following a one hour noise stress session displayed a
reduction in social behaviors and an increase in self-oriented behaviors.

However, many results of social behavior measures did not correspond
to the pattern of results found with cortisol levels. Social behavior measures
are highly dependent upon the context and behavior of the other animals. In
this study social groups were carefully constructed several weeks prior to the
onset of testing, and only one noise stressed animal was in a group at any
time. Nealis and Bowman (1972) found a greater number of significant changes
in playroom behavior following noise stress, but all of the animals in their
playroom groups had been noise stressed at the same time, producing a quite
different behavioral context from the present study.

The effects of control or lack of control over an aversive stimulus on
cortisol levels reported here parallel the results of Weiss (1968) finding
gastrointestinal ulceration in stressed rats and the various performance
measures used in several human studies (Champion, 1950; Gear et al. 1970;
Glass et al. 1971; Staub et al. 1971). Plasma cortisol difference measures
would seem to be of use in evaluating the short-term effects of stress and of
control or lack of control over stress. Cortisol measures might be a more
reliable index of stress in humans than the GSR measurements typically used.

REFERENCES

22, 846-852.

Handler, R. J., Stolar, G. R., and Bern, D. J. (1963). Self-observation as a source of pain
perception. J. Pers. Soc. Psychol. 9, 205-209.

Nealts, P. M., and Bowman, R. E. (1972). The effects of man-made noise on social behaviors and plasma cortisol levels of rhesus monkeys. National Science Foundation (SOS Program) Grant No. 64-9634, Final Report.
### STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tbody>
<tr>
<td>C. S. Harris, H. C. Summer, and D. L. Johnson</td>
<td>Biological Acoustics Branch, 6370th Aerospace Medical Research Lab, Wright-Patterson AFB, OH 45433</td>
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<td>English</td>
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</table>

**Type & duration of experiment:** Not applicable

**Purpose for study:** Review article on effects of infrasound on reaction time, equilibrium, nystagmus, and human performance.

**Description of test groups (subjects, &c., &c.):** Not applicable

**Control of other stresses:** Not applicable

**Statistical Methods:** Not applicable

**Noise Stimulus:**
- Source: sound having alternating, low frequency pressure changes
- Spectral characteristics: infrasound below 16 Hz
- Noise level: 105-120 dB
- Length of exposure: not applicable
- # of trials: not applicable

**CVS Response Measured:**
- Nonauditory effects studied:
  - equilibrium
  - vestibular response
  - nystagmus (involuntary eyeball movements)
  - reaction time
  - performance

**Author's conclusions:**
The effects of infrasound at low intensity levels have been exaggerated or do not exist at all. The level at which infrasound becomes dangerous is still unknown.

**Evaluation & comments:**
Authors try to refute the results of many infrasound studies done on human performance, nystagmus, and subjective response.
Review of the Effects of Infrasound on Man

C. Stanley Harris, Henry C. Sommer, and Daniel L. Johnson

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In recent years, infrasound has been suggested as having effects at much lower intensity levels; indeed, at levels below the threshold of perception. This suggestion is of considerable concern to the Air Force since many maintenance men work with jet aircraft that routinely produce levels up to 130 dB (7).

Some studies on the effects of infrasound on man claim so many dire effects that most investigators will question their credibility. For example, Gavreau (13) states: "...weak infrasounds affect the semicircular canals—the balancing mechanism in the ear—and produce fatigue, dizziness, irritation and nausea. They are certainly one of the many causes of allergies, nervous breakdowns and other unpleasant phenomena of modern life which are found in industrial cities ..." Subsequent to Gavreau's publication (13), the study of infrasound appears slightly more "scientific"; however, many ominous claims have resulted. Authors have suggested that infrasound can: a) make people feel subjectively drunk (4,29), b) adversely affect human performance (21), and c) serve as a useful clinical tool for assessing vestibular function because of its potential for eliciting nystagmus (9).

There are good reasons for questioning the conclusions contained in most studies on the effects of infrasound on man. Most studies are weak in experimental methodology and in scientific reporting. Experimental procedures, statistical methods, and even the number of subjects are often either omitted entirely or so cursorily presented that the reader cannot be sure what was done.

PERFORMANCE MEASURES

Infrasound at levels from 105 dB* to 120 dB has been reported to produce a state akin to alcoholic intoxication in people and to increase their reaction time (10,21). Evans and Tempest (10) state: "It would appear that these psychological effects of infrasound, in the context of transportation, are perhaps more important than the balance, disturbance effects. The subjective

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*Throughout this paper, dB is the R.M.S. sound pressure level referenced to 20 μPa = 20 μN/m².
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descriptions of the subjects as feeling intoxicated are very accurate, since the changes in reaction time are similar to those experienced by a man of average weight who would not pass a breathalyzer test... "Aside from the questionable reasoning, the authors (10) are suggesting that infrasound at higher levels, above 120 dB, adversely affects "balance" by directly stimulating the vestibular system and, at lower levels of 105 dB to 120 dB, producing a state similar to alcohol intoxication through some unknown mechanism. Why adopt two different mechanisms for explaining the effects of infrasound since one of the most sensitive indicators of alcohol ingestion is a disturbance of balance? Fregley et al. (11,12) demonstrated that standing with eyes open on a narrow rail was the most sensitive measure of their staxi task battery to the percent level of alcohol in the blood stream. Therefore, the more interesting question concerning the infrasound studies is why the subjects' reaction times increased, and why they reported feelings of drunkenness at lower intensity levels than were required to adversely affect their balance. Hood, Leventhal and Kyrilaki (21) found a balance test, standing with eyes open on a 1-lb. wide rail, was unaffected by levels of infrasound that they report produced an increase in reaction time.

More important than the discussion of the interpretation of the results is an examination of the results themselves. In three different articles where it is suggested that infrasound at levels of 105 to 120 dB may produce a state similar to alcohol intoxication in the subjects, either no data are presented or it is not obvious that the data support the claims. Moreover, the article by Evans and Tempest (10) warns the transportation industry about possible adverse psychological effects of infrasound without presenting details of procedure, experimental design, statistical analysis or, indeed, the number of subjects used to come up with the conclusion. Yet they state: "No level or frequency of infrasound in the 1-20 Hz region caused any visual disturbance but relatively low levels (115-120 dB) cause a 30-40% increase in reaction time. (It is intended that a more detailed study of this effect will be published in due course.)" The publication of these claims is, at best, premature but, unfortunately, it is also deceiving to present reaction time results in terms of percentage change without presenting the means and standard deviations of the original reaction time measures. Even then the practice is questionable since a very small change in reaction time can result in a considerable percent change.

In another article (21), the investigators purport to show an effect of infrasound on a pointer-following task and a reaction time task. There is only a brief description of the reaction time task: "... a simple single button reaction-time experiment was performed 50 times (21)." One would want to know what stimulus the subjects were reacting to and what the conditions were under which the subjects reacted 50 times. No reference is made to the fact that procedures for conducting simple reaction time experiments are pretty well standardized (37), and details of training, counterbalancing, and manner of stimulus presentation are quite important in conducting a valid experiment. Nevertheless, the authors state (21): "Pointer following and reaction time experiments showed a deterioration in performance in the noisy environment with a significance better than 0.1%." Graphs are presented for individual subjects on the basis of percent increase in error and percent increase in reaction time. This is very poor form since the analyses of variance should not have been conducted using such scores. Nevertheless, these graphs make it seem unlikely that such a high significance level could have been obtained.

It is possible that they could have meant the 10% level? The acceptance of the 10% level is unsatisfactory in the view of the present authors because of the unusual nature of the findings and the small number of seven subjects. The effect reported for reaction time and pointer following was unusual because the percent increase in error and percent increase in reaction time was larger at 110 dB than it was at 120 dB. Although the results for the two sets of scores tend to agree, there is essentially no correlation between the two sets of scores. As one can readily determine by comparing each subject's relative position in both tasks, therefore, one could not predict from the reaction time scores the score a subject would receive on the pointer-following task. This is unusual, since both scores supposedly change because of a state analogous to alcoholic intoxication created in the subject by infrasound.

In discussing the results obtained with a multitasking task, the authors (21) state: "... In the balance experiment only two of the subjects were significantly affected by infrasound..." This conclusion was reached even though an analysis of variance showed no statistically significant effects. At least no effects may have been shown; the authors point out that an analysis of variance was used with the balance test but do not report the results except the above statement that two subjects were "significantly affected." This type of reasoning in interpreting experimental results is misleading and time consuming since it can lead an investigator to discover effects that do not exist. A behavioral measure is not a fixed quantity but is subject to change, not only as a function of the independent variable but also a function of procedural, task, and subject variables as well. The purpose of statistical analysis is to determine whether uncontrolled factors can explain the observed differences.

The statement is made in another article (4) that: "Experiments have shown... that a band of noise 2 Hz-15 Hz wide at a level of 105 dB, or a 7 Hz tone at the same level, can produce an increase in visual reaction time of 10% in half of the subjects in a test group..." Such statements are not informative and no substitute for the use of statistical methods.

A recent, well-designed study by Borredon (2) casts further doubt on the reliability of the above results. He found using 42 young men as subjects, that a 75 Hz infrasound stimulus presented for a 50-min duration at an intensity level of 130 dB had a negligible effect on simple reaction time.

Until evidence is presented in a clearly documented fashion, there is no reason to assume that low levels of

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Infrasound, 120 dB and below, increases reaction time or that infrasound has an effect at low levels that is on a different dimension from that obtained at higher levels.

NYSTAGMUS MEASURES

Another suggestion made by several authors (8-10) is that infrasound stimulates the vestibular receptors and thereby elicits nystagmus. Nystagmus should be a less-variable measure than reaction time; however, when the response is very weak this may not be the case. Nystagmus is subject to many artifacts of measurement, and there are numerous types of nystagmus that are not vestibular in origin. The results in these articles are presented in a case history fashion or there is a simple designation of the number or percentage of subjects showing clear nystagmus, or a slight nystagmus partially masked by random eye movements. No attempt was made to quantify the nystagmus results in terms of eye movement velocity or frequency of beats, and the criterion used to determine the presence of nystagmus was not defined. This could be quite important since, in two figures purporting to show a nystagmus response to infrasound, the nystagmus is quite small compared to that elicited by caloric stimulation and rotational tests (15).

Furthermore, the authors (10) seem to assume that nystagmus should be obtained since they state: "...the majority of observers show some vertical nystagmus under intense infrasonic stimulation, and in fact, examination of eye movement traces indicates that those who did not show a clear nystagmus were, in almost all cases, producing random eye movements sufficient to mask a small nystagmus..." It is also possible, in recording random eye movements, to find sections of the graph paper where the eye movements look like nystagmus. In studies conducted in our laboratory (17) artifacts have often been produced that looked like nystagmus. Control procedures demonstrated that the "nystagmus" was due to eye blinks (and partial eye blinks and squinting), reflexive muscle contractions about the eyes, turning the head, extreme deviation of the eyes (gaze nystagmus), amplifier drift, and, indeed, a combination of many of the above.

The initial assumption that led these investigators to study the efficiency of infrasound in eliciting nystagmus is not based on sound logic. The authors (9), in writing about the effects of low-frequency acoustic signals in the range of 2-20 Hz state: "...This is within the normal frequency range of the semi-circular canals and the upper frequency range of the otolith." This statement is partly true since there is some overlap between the two frequency ranges (26). However, why should there be a relationship between frequency of infrasound, and the frequency sensitivity of the vestibular system to accelerative forces? Certainly, there is no necessary relation. In addition, 7 Hz was reported to be the most sensitive frequency for eliciting nystagmus, and 7 Hz is certainly not within the most sensitive frequency range for either the semicircular canals or the otolith receptors. If there is a relationship between the frequency of infrasound and the frequency sensitivity of the vestibular receptors, one would expect greater sensitivity, more nystagmus, at the lower frequencies below 2 to 4 Hz.

In one article (9) a tracing of "vertical nystagmus induced by a 7 Hz stimulus at 142 dB" is presented as a Fig. 3. The authors do not say whether the eye movement recording was made with eyes open or closed. They state: "Throughout the tests, each experiment was repeated with eyes open and closed." In another article (10) they recorded with eyes open to reduce the possibility of obtaining spontaneous nystagmus. Therefore, one would assume the tracing was obtained with eyes open. However, it is an unusual tracing since there do not seem to be any blinks and the recording is longer than 40 s. Regardless of whether the recording was made with the eyes open or closed, the question remains whether this eye movement record demonstrates nystagmus. The authors state (9) that "an example of nystagmus response in the form of two periods of nystagmus with a few seconds interval between them." If this figure illustrates a typical nystagmus response to infrasound, it is indeed a weak response. In this over 40 s long tracing, only approximately the first 15-s segment shows movements that resemble nystagmus beats. If one scores the six beats occurring just during this period for eye movement velocity in the slow phase, based on the author's calibrations for 20° of eye displacement and for time, the slow phase velocity is approximately 1.5°/s. It would be difficult to classify this response as nystagmus under any system for evaluating nystagmus response (1,20). Such questionable nystagmus might be accepted by some investigators as threshold levels for vestibular stimulation with caloric or rotational stimuli; however, with these stimuli a clearcut nystagmus that is easily scoreable, can be demonstrated at a slightly higher intensity level. Whether this can be demonstrated with infrasound seems questionable. And the fact that these authors, on the basis of their data, propose the use of infrasound as a clinical tool for assessment of vestibular function is truly astonishing. To further complicate this interpretation the authors state (9): "...in the course of the objective (nystagmographic) measurements, no horizontal eye movements were recorded but some 2/3 of the observers showed some evidence of vertical nystagmus, which rather surprisingly, occurred at the end of the stimulus rather than during stimulation." This finding is surprising and so is the statement that no horizontal eye movements were recorded (they probably meant horizontal nystagmus) since Fig. 2 in the same article is titled: "Horizontal eye movements induced by a 10 Hz stimulus at 140 dB (observer 2)." Finally, the authors conclude (9): "...The fact that the eye movements are vertical rather than horizontal suggests that the low frequency effect operated mainly on the otolith system rather than the vestibular system." The otolith system is generally considered to be part of the vestibular system and, however, if by "vestibular system" they are referring to the semicircular canals, then the conclusion is still unwarranted. There are four vertical canals and two horizontal canals, and one may legitimately question whether vertical nystagmus indicates primarily a stimulation of
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the otolith system.

Fig. 2 in a subsequent article (10) is an eye movement tracing of "Vertical nystagmus induced by a 7 Hz stimulus at 130 dB." This figure is "... a typical vertical nystagmus trace recorded with binaural stimulation.--(binural stimulation presented in antiphase at the two ears.)" In this tracing, the nystagmus looks larger and more consistent than in the Fig. 3 referred to in the previous article (9), where subjects were presented with a monaural stimulus; however, part of this is due to the difference in the calibration scale and to the fact that the figure is presented upside down relative to Fig. 3 (in Fig. 2, up is down and vice versa). Turn Fig. 2 over and at least three of the larger nystagmus beats are exaggerated because of from the calibrating eye blink or muscle tension. If one eliminates the first beat in the record, because there is no indication of where the baseline began, and also eliminates the three beats that seem combined with blinks, and corrects for the time in which these eye movements occur, a nystagmus with a slow phase velocity of 1.5°/s or less is again obtained. The authors do not state whether these eye movements were obtained during infrasonic stimulation or after infrasonic stimulation, as they were in the preceding study (9). Nevertheless, the eye movement tracing presented as typical of nystagmus produced by the binaural stimulation with 7 Hz infrasound is unimpressive and open to question as to whether it is a vestibular nystagmus, particularly since the nystagmus response was reported to occur at approximately 10 dB above threshold as indicated by their own threshold curve presented in Fig. 3 (10). Matters of procedure must be clarified before such "low level nystagmus" can be accepted as accurate.

Other forms of "inadequate" stimuli, such as alcohol (1,16) and caloric (5) stimulation, can produce a vigorous nystagmus, whereas the two eye movement tracings of vertical nystagmus in the studies on infrasound (9,10) show the eye velocity to be in the same range as a weak, spontaneous nystagmus. If, according to the threshold curve (10), nystagmus was elicited at 120 dB after 60-70 s, why not show the response at 150 dB after the same period of time. Shouldn't a vigorous nystagmus be produced? Their discussion and threshold curves would lead one to expect at least a quasi-linear relationship (9,10). In a preliminary study conducted in our laboratory (19), no nystagmus was obtained at the 155 dB level, with a 5 min stimulus.

SUBJECTIVE MEASURES

The discussion of the "subjective" effects of infrasound is just as confusing as the discussion of the performance and nystagmus measures. The authors do not tell how the judgments were obtained or how they varied with the intensity of the stimulus. The possibility of suggestion affecting the content of subjective judgment is very real, and particularly in this series of articles on infrasound because of the type of subjective response that was elicited. Evans et al. (9) points out: "... the subjective sensation most commonly reported is one of 'swaying' the apparent movement being away from the ear under stimulation." Howard and Templeton (22) point out that the sway response is very susceptible to suggestion, and increase in sway as a function of suggestion has been proposed as a measure of hypnotizability. These authors (22) were concerned with the sway response and not with subjective sensation of sway; nevertheless, assessment of a person's subjective feelings must be handled meticulously because of the possibility of suggestion affecting the results. Investigators in the area of drug research typically use double-blind procedures and placebo groups because of the difficulty in obtaining valid reports of subjective experiences (28). These methods should prove useful in the study of the effects of infrasound since the stimulus is unique and ambiguous—ambiguous because the subject knows he is participating in an experiment on sound (if he is not told, he can still see the speakers or headphones)—and yet, as has been pointed out (6), the infrasonic stimulus below approximately 14 Hz loses its tonal quality and is experienced as "pulsing" or "throbbing." Furthermore, in discussing subjects' responses to monaural stimulation, Evans, et al. (9) point out: "... although the sound pressure levels required to stimulate a normal person are relatively high, the region of 140 dB, the sensations experienced are not particularly unpleasant." Therefore, how would a cooperative subject respond to a throbbing stimulus that is not unpleasant if he is pressed for a judgment? He might report that he felt swaying. People generally tend to try to make "sense" even out of ambiguous stimuli (see Schultz (33) p. 177 for an interesting summary of the role of set or suggestion in sensory restriction research, and see Geldard (14) p. 416 for a discussion of illusory movements.

DISCUSSION

Regardless of whether performance, nystagmus, or subjective measures are considered, it seems certain that the adverse effects of infrasound reported at low-intensity levels either do not exist or have been exaggerated. In a recent popular article (3), infrasound has been discussed in a more conservative manner than in most of the articles reviewed in the present paper. However, the author emphasizes that there may be "sensitive subjects" who are affected at much lower intensity levels of infrasound than the general population. It would also have been desirable to point out that the burden of proof is on those investigators who claim to have demonstrated effects at very low levels, particularly those who claim to have demonstrated effects at levels below the threshold of perception.

In another recent review, the author makes several suggestions from his review of the literature that the present authors believe are questionable. For example, the author (26) states: "... even fairly low amplitude (in the range of 70 dB) 'may' have physiological effects." And in another part of the paper, he suggests that "vestibular and cochlear dysrhythmia" stem from infrasound in the 110-130 dB range. These suggestions would probably not have been made if the author had also reviewed the results of the "Colloquium on Infrasound" held in Paris, France, September 1973 (24).
REVIEW OF INFRASOUND—HARRIS ET AL.

The question remains concerning the intensity levels of infrasound that can be considered "safe." The United States Environmental Protection Agency, in the document "Public Health and Welfare Criteria for Noise, July 27, 1973," considers levels of infrasound below 130 dB as not constituting a public hazard (32). Studies conducted in our own laboratory (19, 23, 24, 27, 30, 31, 34, 35) as well as the study by Borredon (2) suggest that considerably higher levels than 130 dB are also safe. We have been unable, using human subjects, to elicit nystagmus at intensity levels as low as 155 dB—our data also suggest that the level must be greater than 170 dB in animal subjects—and unable to demonstrate adverse effects on equilibrium at levels to 140 dB (19, 24).

Certainly, more research is needed on the effects of infrasound on man. Duration of exposure and interaction of infrasound with sound in the audible range must be studied systematically. However, a statement by Lipscomb (25) concerning the effects of noise in general is worthy of consideration by anyone undertaking research in the infrasound area: "Be cautious. Many people are willing—yes, eager—to hop onto the 'cry wolf' bandwagon and rant about the grave problems of noise and other contaminants are causing. Careless over-statements can result in unfortunate dilution of factual evidence. Alarmists create a credibility question which fosters contemptuous disregard for new knowledge.

REFERENCES
SECTION 30
**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tbody>
<tr>
<td>B. S. Haskell</td>
<td>Department of Orthodontics</td>
</tr>
<tr>
<td>Eastman Dental Center</td>
<td>University of Pittsburgh</td>
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| Citation                  | Haskell, B. S. Association of aircraft noise stress to periodontal disease in aircrew members. Aviation, Space and Environmental Medicine 46(8):1041-1043, Aug. 1975. |

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<tr>
<th>Type &amp; Duration of Experiment</th>
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<tr>
<td>Short-term field study using x-rays and hearing threshold tests to evaluate groups of aircrew members for periodontal disease</td>
<td>The relationship between aircraft noise exposure and periodontal disease in aircrew members as measured by alveolar bone (the bone that holds the tooth) loss.</td>
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<th>Description of test groups (subjects, age, etc.)</th>
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<td>3 groups of 25 men from the Pennsylvania Air National Guard: Group 1 - jet pilots; Group 2 - pilots and crew of propeller aircraft; Group 3 (controls) - enlisted men not exposed to aircraft noise. (Average age of groups 1 and 2 similar, 8 years younger for controls)</td>
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<th>Statistical Methods</th>
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<td>1 x 2 factorial F test analysis of variance; correlation coefficients</td>
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<th>Noise Stimulus</th>
<th>CVS Response Measured</th>
<th>Nonauditory effects</th>
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<tr>
<td>source: aircraft noise</td>
<td>Alveolar bone loss using full-mouth x-rays - the more flight hours, the more alveolar bone loss in propeller crew; propeller crew suffered greater bone loss than jet crew.</td>
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<tr>
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<td>noise level: 94-118 dBA (various stages of flight)</td>
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<tr>
<td>length of exposure: 1000 - 4000 flight hrs.</td>
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<td>n of trials: not applicable</td>
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<table>
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<tr>
<th>Author's conclusions</th>
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<tbody>
<tr>
<td>Alveolar bone loss was much greater in propeller crew members than in jet crew members, which was only slightly higher than in the controls with no aircraft noise exposure. Flight hours were correlated to hearing loss in both jet and propeller crew members. The difference may be due to the greater vibration associated with propeller aircraft.</td>
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<tr>
<th>Evaluation &amp; comments</th>
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<tbody>
<tr>
<td>1) More controlled laboratory studies are needed to understand this phenomenon.</td>
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<tr>
<td>2) The age of the subjects were not specified.</td>
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Association of Aircraft Noise Stress to Periodontal Disease in Aircrew Members

BRUCE S. HASSELL
Department of Anthropology, University of Pittsburgh, Pittsburgh, Pennsylvania 15260


A review of the literature reveals a multitude of effects that noise may contribute to periodontal disease, including cardiovascular disease, angina pectoris of peripheral vessels, hypertension, and an increase in inflammatory cells with concurrent inhibition of lesion. Three groups of 25 men were selected from the Pennsylvania Air National Guard for study. Group 1 consisted of F-102A jet fighter pilots; Group 2, pilots and crew of a four-engine, propeller-driven C-121 aircraft; and Group 3, enlisted men not exposed to aircraft noise, as a control. The degree of parallel, intracapsular bone loss for each subject was measured on full-mouth radiographs of all groups. The greatest amount of bone loss occurred in crew members of propeller-driven aircraft. A pilot had considerably less bone loss while the range number of millimeters of bone loss per tooth revealed differences between the three groups at the 0.01 significance level (F=4.47). The data suggests there is a degree of arteriosclerotic bone loss over a period of years associated with exposure to aerodynamic noise and vibration, and negligible loss for aircraft noise.

For many years it was believed that periodontal disease (pyorrhea) was caused largely by local irritation and mechanical factors, and was treated with some success in many of the cases. However, the disease curtailed or persisted in many of the patients treated. It is now known that periodontal disease is the result of a complex of both local and systemic factors with no single etiology. There have been reports that psychosomatic factors defined as "anxiety" or "stress" often give rise to definite pathological processes in the periodontal structure. In this paper, the subject of noise "stress" as a factor in periodontal disease will be considered.

The autonomic nervous system exerts control over the oral supply and, thereby, the nutrition for all parts of the body. It is known that blood vessel adventitia contracts in an exaggerated fashion and may remain contracted for extended periods of time from emotional stress. It is also possible that periodontal pathology could occur as a physical response to an inadequate supply of oxygen and other nutrients or a continued blood vessel constriction.

Manhold (12), summarizing the previous reports on tissue metabolism, reported that gingival tissue respires at a normal rate commensurate with its state of health or disease. Under normal circumstances, increased activity on the part of tissue requires an increased quantity of oxygen and other nutrients. This is provided by (1) abstraction of a larger amount of oxygen from any given blood volume by elevation of the oxygen utilization coefficient, and (2) an increase of total blood flow to the tissue. The total blood flow can be increased by dilation of the blood vessels and opening new capillaries to the area.

If incipient periodontal breakdown were precipitated, as in oral neglect, the reparative process might not be adequately initiated as a result of stress. In this case, the total blood flow increase to the affected area might not occur because of interference with formation of new vessels, with vessels already dilated, or with any repair process previously underway.

Noise might contribute a multitude of effects to periodontal disease. Evidence for this may be summarized as follows:

1) Periodontal pathology and the weight of the sympathetics in experimental animals increased with auditory and other stress (9-11, 13).

2) People professionally exposed to noise are subjected to cardiovascular stress severe enough to cause pathology (14).

3) Adolescents have a tendency to develop angiospasms of the peripheral vessels due to noise and vibration (16).

4) There is more arterial hypertension among workers subjected to noise than among office workers or manual laborers (15).

5) The reaction of bodily defense to inflammation in animals is partly inhibited with auditory stress (17, 18).

6) There is an increase of inflammatory cells in the blood with noise stress (19).

7) Anxious and mentally disturbed people are more sensitive to noise, and this group as a whole is more liable to have periodontal disease (1-8).

This study was prepared under the auspices of the Public Health Service Training Program, Grant #5A67 AI 00251.

The present address of the author is Department of Orthodontics, Eastman Dental Center, Rochester, NY 14603.
that the more hours aloft, the more alveolar bone loss was present, regardless of type of aircraft flown.

In Table V, the means for alveolar bone loss demonstrate that officers in the propeller group had the greatest bone loss $\bar{x}=1.22$, compared with the jet groups $\bar{x}=0.39$. Officers in the propeller group with over 2400 h of flight time had the greatest bone loss, $\bar{x}=1.55$. A T test conducted between the propeller group and the control group for bone loss also proved significant to the <0.05 level of confidence.

**DISCUSSION**

It is not surprising that there are similarly high correlations between flight hours and hearing thresholds of $r=0.67$ and $r=0.70$ for both Groups 1 and 2, respectively. However, it seems unusual that the correlation between flight hours and mm/T is negligible for Group 1, and so significant for Group 2. The analysis of variance also clearly indicates a significantly high amount of bone loss in propeller aircraft pilots with a high number of flight hours.

The noise level that jet pilots experience is negligible during flight, yet severe on the flight line, while the propeller-driven aircraft pilots and crew are exposed to intense noise in both situations. An explanation for the comparatively large amount of alveolar bone loss for the propeller pilots, while the correlations between flight hours and hearing threshold for both Groups 1 and 2 are nearly identical, may lie in the extreme difference in the type of noise and vibration for the two types of aircraft. An analogy might be drawn to Teyssar’s work, in which subjects worked with jackhammers and other noisy and vibratory low-frequency construction instruments (15). In this group, a tendency for the development of angioplasia of the peripheral vessels was experienced. Another factor may be the difference in in-flight atmospheric pressure between jet and propeller aircraft.

Although aging plays a large role in the amount of alveolar bone loss, the average ages for Groups 1 and 2 are only 1 year apart. Group 3, whose average age is approximately 8 years younger than the other groups, has a significantly lower mm/T score.

The importance of the role of psychological stress has already been reported. It is not known if there is inherently more stress in flying a large multiengine aircraft with a crew and dozens of passengers, than in flying the single-seat jet aircraft, but this too may be a factor.

It is assumed that there is no difference in the type of person who becomes a jet or propeller pilot, since the choice is made by the Air National Guard strictly on the basis of need. A pilot may be asked to switch from one type of aircraft to another when accepting a position with the Guard.

**ACKNOWLEDGEMENT**

I would like to express my gratitude to my supervisor, Charles W. Dean and Edmund M. Rice, and to other members of the Graduate School of Public Health, Social Science Unit, for the helpful suggestions they offered. I would also like to thank Col. Paul Rosenberg of the Pennsylvania Air National Guard for permitting me to examine the health records of his men.

**REFERENCES**

AIRCRAFT NOISE & PYORRHEA—HASKELL

MATERIALS AND METHODS

In order to test the possibility that noise stress contributes to periodontal bone loss in humans, an experiment was devised to ascertain the degree of alveolar bone loss in persons subjected to aviation noise stress. Three groups of 25 men with complete medical and dental records were selected from the Pennsylvania Air National Guard. Group 1 consisted of F-102A jet fighter pilots; Group 2, of pilots and flight crew of the four-engine, propeller-driven C-121 aircraft; and Group 3, of enlisted office workers not exposed to aircraft noise, as a control. In order to determine the total amount of noise exposure for Groups 1, 2 and 3, the number of hours of flight time was noted for each man.

In Group 1, the jet pilots wear custom-fitted helmets, specially equipped with ear protectors, thereby making cockpit noise in flight negligible. While on the ground at the flight line, these pilots may be exposed to extreme noise, usually without the benefit of the protective flight helmets. It was only 6 months prior to this study that a new directive went into effect requiring ear plugs or a helmet to be worn at the flight line.

In Group 2, the flight crew is without protective headgear and is continually exposed to noise on the ground and while flying.

Current full-mouth radiographs, of all three groups, taken with the prescribed USAF Dental Corps technique, were examined on a standard radiograph viewbox. The degree of existing bone loss was charted and measured with a bone gauge caliper to the nearest 0.1 mm. The total number of millimeters of bone loss was then divided by the total number of existing teeth, thus giving each individual a score of the average number of millimeters of alveolar bone lost per tooth (mm/T). These scores represent an underestimate in all films because it is impossible to measure bone height on the buccal and lingual surface of each tooth in the radiographs.

Hearing threshold tests, performed at the Maltese audiometer at the ISO setting, were also recorded for all groups. (ISO: International Standard Organization (1964) F = 1000 Hz 0.6-5 dB re 0.0002 dyn/cm²). Since prolonged noise is known to damage hearing, it seemed reasonable to compare the easily identifiable parameter of hearing threshold to the amount of flight hours, thereby indicating a measurable degree of hearing loss for Groups 1 and 2. Hearing loss was then compared for each individual to the number of flight hours and with the increase of alveolar bone loss to determine possible associations. An average was calculated for the hearing threshold of right and left ears, at a frequency of 4,000-6,000 Hz.

RESULTS

The levels of sound in decibels measured with a sound level meter for Groups 1 and 2 are given in Table I. The present Federal Standard is 90 dBA for an 8-h exposure. The averages of flight hours, mm/T, hearing threshold, and age for all three groups are given in Table I.

An F test analysis of variance revealed a difference between the three groups at the 0.01 level of significance for both average mm/T (F = 24.7) and average hearing loss in decibels (F = 7.54).

<table>
<thead>
<tr>
<th>Flight Line*</th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aircraft at idle</td>
<td>105</td>
<td>94</td>
</tr>
<tr>
<td>Maintenance generation</td>
<td>101</td>
<td>102</td>
</tr>
<tr>
<td>and other support equipment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In-Flight**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Takeoff</td>
<td>Negligible</td>
<td>118</td>
</tr>
<tr>
<td>Climb</td>
<td>=</td>
<td>116</td>
</tr>
<tr>
<td>Level Flight</td>
<td>=</td>
<td>113</td>
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</table>

*Flight line conditions to which all pilots have been exposed without mandatory ear protection.
**In-flight ear protection maintained only by jet pilots.

<table>
<thead>
<tr>
<th>Flight Hours/ (mm/T)</th>
<th>Flight Hours/ (Hearing Threshold)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Jet</td>
<td>1.5 = 0.29</td>
</tr>
<tr>
<td>2 Propeller</td>
<td>r = 0.73</td>
</tr>
<tr>
<td>3 Control</td>
<td>no flight hours</td>
</tr>
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</table>

- Pearson's r correlation coefficient = significant correlation.

<table>
<thead>
<tr>
<th>Source of Variation</th>
<th>df</th>
<th>Sum of Squares</th>
<th>Mean Squares</th>
<th>F Ratio</th>
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<tbody>
<tr>
<td>Hours in Flight Time</td>
<td>1</td>
<td>7.59</td>
<td>7.59</td>
<td>36.14*</td>
</tr>
<tr>
<td>Jet-Prop</td>
<td>1</td>
<td>8.83</td>
<td>8.83</td>
<td>42.04*</td>
</tr>
<tr>
<td>Interaction</td>
<td>9</td>
<td>4.06</td>
<td>4.06</td>
<td>19.33*</td>
</tr>
<tr>
<td>Error</td>
<td>44</td>
<td>9.49</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>47</td>
<td>29.97</td>
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* = significant at < 0.01

Correlations between flight hours and mm/T, and between flight hours and hearing thresholds for Groups 1 and 2 are given in Table III. This analysis indicated there was no significant correlations in Group 1 between flight hours and mm/T, while all the other correlations indicated are significant to the 0.01 level of confidence.

In Table IV, the results of a 2 x 2 factorial analysis of variance indicate that there are significant interaction effects between the number of flight hours and the type of plane. An examination of the flight times also indicated.
### Summary Form for
### Studies on the Effects of Noise on the Cardiovascular System (CVS)

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<td>F. M. Nalis</td>
<td>Institution and address where research was performed</td>
</tr>
<tr>
<td>E. E. Bowman, Ph.D.</td>
<td>University of Wisconsin, Primate Laboratory</td>
</tr>
<tr>
<td></td>
<td>22 N. Charter Street</td>
</tr>
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### Citation
Nalis, F. M. and Bowman, E. E. Behavioral and corticosterone responses of rhesus monkeys to noise-induced stress... (unpublished paper).

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<th># of Fig.'s 3</th>
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### Purpose for Study
The effect of different types of noises and varying exposures on corticosterone response and behavior of rhesus monkeys.

### Description of Test Groups (Subjects)
- 16 wild adolescent rhesus monkeys (8 males, 8 females).
- 3 test groups - 2 males, 2 females each - 3 noise types presented randomly for each.
- 1 control group - 2 males, 2 females - no noise.

### Control of Other Stressors
Laboratory conditions - in sound-proof chamber; visual & auditory contact with 3 other subjects in each group, but no tactile contact.

### Noise Stimulus
Source: continuous, variable, or impulse tape.
Recorded noise spectral characteristics: frequency varied with variable noise (music).
Noise level: average level = 100 dBA.
Length of exposure: 1, 2, and 3 hrs. (2 30-min. recordings of each noise type).
No of trials: unspecified.

### Statistical Methods
- 3 x 3 factorial design; f-tests; analysis of variance.

### CVR Response Measured
- None.

### Nonauditory Effects
- Plasma cortisol level - initial rise during first hour of noise exposure; normal after 3 and 5 hrs. of noise.
- Behavior - greater fatigue due to noise after 3 hrs. continuous or variable noise or after 5 hrs. impulse noise.

### Author's Conclusions
Corticosterone levels were elevated significantly after one hour of noise exposure but not after 3 or 5 hrs. This indicates an adaptation after 3 and 5 hours of noise. Type of noise did not affect the plasma cortisol response, but did affect behavioral response.

### Evaluation & Comments
Plasma cortisol responses to noise seem to be similar in monkeys and humans. Behavioral responses would be more difficult to compare.
Behavioral and Corticosteroid Responses of Rhesus Monkeys to Noise-Induced Stress

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Madison, Wisconsin 53706

Adrenal and Behavioral Responses to Noise
Abstract

Adolescent rhesus monkeys were exposed in randomized block order to 1, 3, and 5 hr sessions of continuous, variable, and impulse noise. As an index of physiological trauma, plasma cortisol concentration was measured immediately at the termination of each auditory exposure. Immediately thereafter behavioral and social activities of the monkeys were charted by a modified frequency counting technique. At the termination of all 1 hr noise exposures, plasma cortisol levels were elevated to 80 per cent of maximum rise, but had returned to control levels at the termination of all 3 and 5 hr noise exposures: After 1 and 5 hr exposures to continuous and variable noises, the monkeys generally exhibited control levels of behavior, but after 3 hr exposures they showed reduced behavioral activity with corresponding increases in nonsocial behavior, especially sleep. In contrast, this behavioral alteration was not induced by impulse noise after 1 and 3 hr exposures, but was seen after 5 hr exposures. These findings suggest extra-auditory effects of noise in reducing arousal or in producing a general fatigue that is manifest in a "free response" social situation.

Corticosteroids Noise Rhesus Monkey Social Behavior Stress
Behavioral and Corticosteroid Responses of Rhesus Monkeys to Noise-Induced Stress

The prevalence of noise in residential areas, places of public use, and work situations has generated considerable concern about the possible effects on man of this kind of sensory stimulation. A well-documented effect of exposure to intense noise is hearing loss manifested as either a transient or a permanent shift in auditory sensitivity thresholds. However, the extra-auditory effects of noise are not well understood (see Leake, 1970). Research has concentrated mainly on the effects of noise on task performance by human subjects, although the methods and findings of such experimentation remain somewhat controversial (Broadbent, 1957; Kryter, 1966). Moreover, noise-induced changes in task performance offer little generalization to other important aspects of human behavior. In particular, the possible effects of noise on social behavior have been largely unexplored. A major objective of the present study, therefore, was to delineate some of the consequences of prolonged noise exposure in regard to the behavioral responses of a primate species. The rhesus monkey was employed since it has often proved to be an appropriate species for modeling human behavior.

Emotional states in humans following noise exposure have been extensively investigated by the self-report method (e.g., Kryter, 1966; Laird, 1929; Laird and Cove, 1933). For research on the monkey, there seems to be no objective index of emotionality that embodies specificity as to the quality or intensity of the sensation produced. However, considerable evidence exists to suggest that the activity of the pituitary-adrenal cortical system provides
a reliable measure of physiological and psychological trauma induced by environmental stressors (Bohus, 1968; Mason, 1968; Meyer and Bowman, 1972). A few investigators have, in fact, used corticosterone levels as indices of noise-induced stress in both humans (Arguelles, Ibeas, Ottone, and Chekherdemian, 1962) and rats (Henkin and Knigge, 1963). The general characteristics of the pituitary-adrenal system of the monkey are well documented as well as the response of this system to psychologically stressful conditions (Mason, 1968; Meyer and Bowman, 1972). Consequently, it was thought that total plasma cortisol concentration could be an effective measure of physiological trauma in rhesus monkeys following noise stimulation.

The physical characteristics of noise are apparently related to certain deleterious effects on the organism (Kryter, 1950; Laird, 1929; Lindquist, Neff, and Schuknecht, 1954; Reese and Kryter, 1944). Apart from loudness, the method of presentation appears to be a critical factor since steady-state noise is less disturbing to humans than intermittent sounds (Plutchik, 1959; Smith, 1951). These findings come from noise studies that employed unusual stimuli such as clicks and white noise presented at extremely high amplitude, e.g., 120-160 dB. In the present experiment, however, noises commonly found in human situations, e.g., in the operation of power tools, were presented at an intensity level that would be common to such situations. It was hypothesized that monkeys would require more time to adapt to intermittent noise than to constant noise, and that increases in noise exposure duration would produce increases in the severity of any deleterious effects observed.
Method

Subjects

Sixteen feral adolescent rhesus monkeys (Macaca mulatta), 8 males and 8 females, served as subjects. To facilitate the scheduling of treatments and observations, the monkeys were assigned to 4 groups of 4 subjects. Pairs of subjects within each group were housed together in wire cages throughout the study with feeding and maintenance schedules closely controlled.

Apparatus

An Ampex model AG600 monaural tape recorder was used to record and play back auditory stimuli. A Neumann omnidirectional condenser microphone provided acoustic pickup while reproduction of the sound was accomplished with an Altec model 1594A amplifier and Electrovoice Sentry IV speaker system (frequency response of 50-20,000 Hz). Noise treatments were administered in a sound-proofed chamber (1.8 X 1.8 X 7 m) that served to attenuate external incidental noise by 30 dBA. A previously described playroom (Harlow, Rowland, and Griffin, 1964) was utilized during pre- and post-treatment behavioral tests.

Auditory Stimuli

Three types of noise stimulation were employed: continuous (C), variable (V), and impulse (I). Two 30 min. recordings of each noise type were produced on magnetic tape at 7.5 ips. In Condition C the noise consisted of continuous sounds of power tools (21 min) and land vehicles (9 min) with the total time for each distributed randomly over the 30 min recordings. The noise for Condition V consisted of "rock" music of the type currently popular; both instrumental and instrumental-vocal arrangements (15 min each) were randomly distributed throughout a recording. The amplitude and frequency of V noise, unlike the steady-state characteristics of C noise, tended
to vary within any given segment because of the nature of music. The I condition was a random collection of shotgun blasts, pistol shots, and bursts of machine gun fire. The occurrence of any one of these sounds was at random. The total noise time in Condition I was approximately 2 min per 30 min recording in contrast to conditions C and V in which noise was present for the entire 30 min period.

Design and Procedure

A 3 x 3 factorial design with repeated measures was employed that consisted of 1, 3, and 5 hr exposures to C, V, and I noise presented at an average amplitude of 100 dBA. Three groups of monkeys were assigned to the noise conditions with the order of treatments randomized differently for each group. The remaining group of four monkeys served as a no-noise control. This latter group followed the same experimental procedure as the other three groups except that 3 no-noise sessions were given at each of the 3 durations. The experimental conditions were administered to all groups over a period of 36 days with treatments separated by a minimum of 90 hr. The monkeys were treated and tested in their respective groups throughout the study and also during 36 days of pre-experimental sessions that were designed to facilitate acclimation to test procedures. The subjects were caged individually during confinement to the sound-proofed chamber and had visual and auditory, but no tactual, contact with other members of their group. Noise and no-noise sessions commenced at different times, depending on the duration of each, but always ended at 15:15 hr.

Cortisol Assay. Plasma cortisol concentrations were monitored throughout the study. A blood sample of 0.4 ml of saphenous or femoral blood was taken with a dried, heparinized syringe at 15:15 hr on the day prior to each
noise or no-noise treatment. Blood samples were also collected immediately after noise or no-noise treatments, i.e. at 15:15 hr. All blood samples were centrifuged immediately, four 10\(\mu\)l plasma aliquots taken, each diluted with 190 \(\mu\)l of distilled, deionized water, and frozen at -15\(^\circ\)C until assayed blind in duplicate by protein binding radioassay for cortisol content (Bowman and De Luna, 1969). An assessment was also made of each monkey's maximal cortisol response to an intramuscular injection of 16 units/kg of ACTH (Armour Acthar), a method that results in maximal stimulation of the adrenal cortex in rhesus monkeys (Bowman and Wolf, 1969). Blood samples were collected at 0, 1, 3, and 5 hr post injection and assayed in the manner just described.

**Behavioral Test.** Noise-induced changes in the behavioral activity and social interactions of the monkeys were charted by a modified frequency counting technique (Suomi, Harlow, and Kimball, 1971) that consisted of 45 consecutive 20-sec intervals per subject/session. The behavioral taxonomy reported in this study is presented in Table 1. Interobserver reliability reached or exceeded the 0.95 level by the product moment correlation method for each behavioral category. A pre-treatment observation period commenced at 9:15 hr and a post-treatment observation period began at 15:45 hr on each day of noise and no-noise treatments. The monkeys were given 15 min to acclimate to the playroom before testing began.

**Data Analysis.** All data were converted to differences between pre- and post-treatment measures in order to assess changes due to treatments. These pre-post differences are termed "change scores" in this report. The change scores exhibited by the no-noise control monkeys under the various exposure durations were assumed to reflect the effects of no-noise experimental procedures such as confinement to the treatment chamber, handling, and venipuncture. Therefore the change scores of noise-treated subjects for each
exposure condition were corrected by subtraction of the mean change score exhibited by no-noise control subjects at the corresponding exposure duration. In this regard, it should be noted that analysis of variance revealed no significant effects of exposure duration on the change scores of no-noise controls for any of the dependent measures reported here. Under the null hypothesis of no effect of noise, the expected value, $\mu$, of these corrected change scores would be zero. The corrected change scores for each dependent variable were then subjected to analysis of variance with planned contrasts.

Insert Table 1 about here

For significant $F$'s (linear) at $\alpha = .05$, subsequent orthogonal $F$ tests were done on the differences of the mean corrected change scores from the null hypothesis value of zero.

Results

Cortisol Data. The monkeys' cortisol responses did not appear to be differentially affected either by the type of noise stimulation or by the interaction of noise type and exposure duration ($F$'s $< 1$), although the main effect of exposure duration was statistically significant ($F = 24.4$, $df = 1/11$, $p < .0005$). An elevation in plasma cortisol concentration was observed at the end of 1 hr noise exposures, but no elevation was seen after 3 and 5 hr noise exposures. The mean changes in plasma cortisol concentration of noise-treated monkeys, corrected by the mean changes exhibited by control monkeys, are shown in Fig. 1 as a function of exposure duration.
When these changes in plasma cortisol levels were compared with maximal plasma cortisol responses to injection of ACTH, the noise stimuli did not appear to be maximal stressors. The activity of the pituitary-adrenocortical system was at 80 per cent of maximum following 1 hr noise exposures (45 µg% vs 57 µg%). After 3 and 5 hr exposures to noise, the monkeys exhibited plasma cortisol concentrations of 40 per cent (35 µg% vs 86 µg%) and 33 per cent (32 µg% vs 96 µg%) of maximal response, respectively, which approximates control levels of plasma cortisol under no-noise conditions. Only the plasma cortisol concentrations following 1 hr noise exposures reached significance at the 99 per cent confidence level. It can be noted that the control monkeys showed nonsignificant pre- to post-treatment increases in plasma cortisol concentrations of 4, 5, and 6.5 µg% after 1, 3, and 5 hr no-noise exposures, respectively.

**Behavioral Data.** The following behavioral categories showed pre- and post-treatment frequencies that were too low to justify analysis: active disturbance, aggression, and submission. The occurrence of locomotion and exploration was of higher frequency, but yielded nonsignificant F ratios (all p's > .08). However, corrected change scores for dependent measures of sleep \( F = 8.0, \text{df} = 1/11, p < .02 \), passive disturbance \( F = 4.8, \text{df} = 1/11, p < .05 \), social contact \( F = 7.9, \text{df} = 1/11, p < .02 \), and social play \( F = 5.2, \text{df} = 1/11, p < .05 \) were statistically significant in the analysis of the interaction of noise type and noise exposure duration.
After 1 hr exposures to C noise, the monkeys exhibited significant increases in passive disturbance and social contact, although levels of sleep and social play remained unchanged (see Fig. 2). However, after 3 hr exposures to C noise, significant increases in sleep were observed and were accompanied by significant decreases in both social play and social contact. Following 5 hr exposures to C noise, the monkeys exhibited significant increases in social play, but other behaviors remained at normal levels.

One-hour exposures to V noise resulted in significant decreases in sleep and significant increases in social play (see Fig. 2). This play behavior appeared to be contact-type play as reflected by significant increases in social contact for 1 hr exposures to V noise. After 3 hr exposures to V noise, both disturbance activity and sleep were significantly increased and social play was significantly decreased. After 5 hr exposures to V noise, the monkeys exhibited baseline levels in all behavioral categories except social play which remained at subnormal levels.

All behaviors remained unchanged following 1 hr and 3 hr exposures to I noise (see Fig. 2). Following 5 hr exposures to I noise, however, significant increases in sleep and passive disturbance were observed, accompanied by significant increases in social contact, and no change in social play.

Discussion

The present data indicate that the pituitary-adrenocortical response to continuous, variable, or impulse noise was similar to that of humans (Arguelles et al., 1962) and lower mammals (Henkin and Knigge, 1963). There was an initial rise in plasma cortisol concentration during the first hour of noise exposure, but an adaptation to normal cortisol levels after 3 and 5 hr
noise exposures. This suggests an initial stress or fear followed quickly by an emotional adaptation.

By contrast, the social behavioral data indicate a more complex picture, relatively uncorrelated with the adrenocortical response. The generalization most apparent in these data was that noise produced a soporific or fatiguing effect on social behavior immediately following certain durations of exposure to the different noises. For example, continuous machine noise produced increased passive disturbance after 1 hr of exposure and increased sleep after 3 hr of exposure; the monkeys then appeared to adapt or recover following 5 hr exposures, showing normal sleep, passive disturbance, and social contact levels, but also showing elevated social play. By contrast, variable noise (music) produced increased social play after 1 hr of exposure, very heightened sleep and passive disturbance after 3 hr of exposure, and recovery to nearly normal behavioral levels after 5 hr of exposure. Finally, impulse noise (gunshots) produced no measured effects on subsequent social behavior after 1 hr and 3 hr exposures, but very heightened sleep, social contact, and passive disturbance after 5 hr of exposure.

Within the exposure durations tested, the monkeys exhibited both onset of and recovery from the soporific effect of both continuous and variable noise, and a more delayed onset of a soporific effect after the longest exposure to impulse noise. Unfortunately, these data in themselves offer little guide to prediction of the effects of longer durations of exposure to noise, or of longer sequences of repetition of exposures. However, these data do raise the possibility that one cost of noise exposures in the human environment may be a fatigue effect that will depress free responding such as that seen in social situations during periods immediately following the noise exposures.
The present data further clarify the monkey's pituitary-adrenocortical response to noise stressors. Rhesus monkeys, like humans (Arguelles et al., 1962) and lower mammals (Henkin and Knigge, 1963), show initial high rates of cortisol secretion in response to noise exposure, but this response is attenuated by further exposures to the stressor. However, the corticosteroid response shows no differentiation among different types of noise. Contrariwise, the behavioral data suggest a parameter of noise effects that seems sensitive to the nature of the noise stressor. Whether or not the behavioral responses of a nonhuman primate species to noise stimulation generalize to the human situation is purely speculative at this time. Perhaps cognitive mediating factors play an important role in the human's struggle to adapt to unusual noise conditions as it appears might be the case in rhesus monkeys (Hanson, Larson, and Snowdon, 1976). Little is known about this factor except that humans show reduced tolerance for frustration under noise conditions, but to a lesser degree when given the impression that they can exercise control over the noise stressor (Glass, Singer, and Friedman, 1969). Although the study of human behavioral reactions to noise is a difficult area of investigation, it may be of significance to determine how man socially behaves following exposure to noise, particularly in light of the present findings.
Footnotes

1. This research was supported by NSF Grant GY-9634 to the first author, and by USPHS Grants MH-11894 and RR-00167 to the University of Wisconsin Primate Laboratory and Regional Primate Research Center, respectively. The authors wish to thank R. De Luna, R. Goldschmidt, J. Klann, E. Hoffman, P. Lehninger, M. Levinson, P. O'Neill, P. Plubell, C. Polnazcek, and M. Walker for their efforts in carrying out the experiment.

2. Reprint requests should be addressed to Robert E. Bowman, University of Wisconsin Primate Laboratory, 22 N. Charter Street, Madison, Wisconsin 53706.
Figure Captions

1. Mean changes in total plasma cortisol concentrations as a function of noise exposure duration. No change with respect to control levels is represented by zero on the ordinate. The 99 per cent confidence interval is shown as the shaded area.

2. Mean changes in the frequency of four behavioral measures as a function of continuous noise (C) vs variable noise (V) vs impulse noise (I) and exposure duration. No change with respect to control levels is represented by zero on the ordinate. The 99 per cent confidence intervals are shown as shaded areas.
Table 1
Behavioral Taxonomy Employed in Playroom Testing

Social Behavior

Social Play: any socially directed play activity, including rough-and-tumble and noncontact play.
Social Contact: tactual contact with another subject exclusive of aggression, grooming, and play.
Aggression: hair pulling, biting, and/or facial threat (an expression consisting of ears pulled back with lower jaw pulled down and out).
Submission: fear grimace (facial expression resembling a smile, produced by pulling the lips back to expose teeth) and/or withdrawal from, or sexually presenting to, an aggressor.

Nonsocial Behavior

Locomotion and Exploration: ambulation of one or more full steps and/or visual, oral or tactual exploration/ manipulation of environmental objects.
Sleep: resting in a sitting or prone position with head lowered and eyes closed.
Active Disturbance: self-mouth (oral contact, excluding discrete biting, with any part of the body), self-bite (specific, vigorous, self-directed bite), rock (repetitive, nonlocomotive forward and backward movement), or stereotypy (identical movements, rhythmic and repetitive, maintained for at least three cycles).
Passive Disturbance: self-clasp (manual or pedal clutching of any part of the body) and/or huddle (self-enclosed "fetal" position with head lowered beyond shoulder level, including any other patterns of self-clasp and self-embrace).
References


SECTION 32
### SUMMARY FORM FOR

**STUDIES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)**

<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lt. Col. Prof. G. Paolucci, LAF, NC</td>
<td>Centro di Studi e Ricerche di Medicina Aeronautica e Spaziale; Via F. Cobetti, 2/a 00185; Roma (Italy)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Investigator's Phone No.</th>
<th>Spurring Organization</th>
</tr>
</thead>
</table>


<table>
<thead>
<tr>
<th>Type</th>
<th>Duration of experiment</th>
<th>Purpose for study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Field, short-term (1.5 - 5 hr.)</td>
<td>To examine the effects of two different high intensity noises (continuous and intermittent) on urinary catecholamine excretion.</td>
<td></td>
</tr>
</tbody>
</table>

#### Description of test groups (subjects, etc.)
20 Air Force ground specialists usually employed in noisy tasks (wearing ear plugs) divided into two test groups of 10 each: Group A - continuous workday-noise of 120 dB for 1.5 hrs. Group B - intermittent impulse noises of 80 - 100 dB for 5 hrs. (Each subject served as his own control - catecholamines tested on nonworking days for control values)

<table>
<thead>
<tr>
<th>Control of other stressors</th>
<th>Statistical Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>no control</td>
<td>not given</td>
</tr>
</tbody>
</table>

#### Noise Stimulus

- source: Group A - jet engine noise
  - Group B - jet takeoff runway noise
- spectral characteristics: not specified
- noise level: Group A - 120 dB continuous
  - Group B - 80 - 100 dB intermittent
- length of exposure: Group A - 1.5 hrs.
  - Group B - 5 hrs. intermittent impulse (a few sec.) noise every 20 min.

#### CVS Response Measured
- Nonauditory effects
  - urinary catecholamine levels - no significant increases after a few hours noise exposure (no adrenal gland response); the levels were all within a normal range

<table>
<thead>
<tr>
<th>Author's conclusions</th>
<th>Evaluation &amp; comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catecholamines did not increase significantly as a result of noise exposure for a few hours, indicating lack of adrenal gland response. Lack of response may be due to ear protection and habituation to noisy work environment.</td>
<td>The inclusion of a control group not normally exposed to noise would have been useful. The age and length of aviation service of the subjects was unspecified.</td>
</tr>
</tbody>
</table>
"INFLUENCE OF THE NOISE ON CATECHOLAMINE EXCRETION"
by
Lc-Col. Prof. G. PAOLucci, IAF, MD
Centro di Studi e Ricerche di Medicina Aeronautica e Spaziale
Via P. Cobetti, 2/a
00195 ROMA (Italy)

N76-17795

SUMMARY

Aim of the work was to know whether a few hours exposure to hazardous noises could not act as a stressing factor and so able to give increase on catecholamines excretion.

The study was carried out on aviation specialists, daily exposed to high noises, fully protected against hearing damage by ear plugs; the exposed people were divided in two groups of ten subjects, each one exposed to different noisy conditions:

- the one was exposed to continuous and steady noise of 120 dB for 1 hour and 1/2;
- the other to intermittent noises of 50-100 dB for 2 hours, with intervals between impulses bursts of 30', lasting each one only a few seconds.

The subjective tolerance was good and no disturbance or fatigue reactions appeared at the end of the exposure. Urinary catecholamines excretion was assayed the day before the test (in noiseless plane) and the next one at the end of the exposure.

As the test values, compared with the blank, showed no changes in catecholamines release, it can be argued that, upon trained people, with hearing fully protected, noise might not act as a conventional stressor, at least at the same conditions of the present research.

INTRODUCTION AND AIM OF THE WORK

The exposure to high levels of noise for a certain duration, apart the auditory effects, can lead to a loss of working efficiency.

The damages of noise upon eardrum and hearing organ are not be taken into consideration in this paper, in which only endocrinological aspects are to.

Since the long exposure to noises, in most people can produce headaches, drop on attention, loss of resistance, we intended to establish whether the noise could act as a stressing factor, able to give rise to increased output of catecholamines from adrenal gland.

METHOD

On 20 IAF ground specialists, usually employed in noisy tasks (wearing ear plugs during the job), this research was carried out. The people were divided in 2 groups of 10 persons, unhomogeneous for age and body size, which were exposed for different durations to different high noise conditions.

In particular, the exposure was executed in the following way in two different places:

- "A" - "engine test area": the workers remained exposed for 1 hour and 1/2; the noise was continuous and steady at level of about 120 dB;
- "B" - "take-off runway": the workers' exposure lasted 5 hours, but the noise was intermittent and lower; it arised at every 1-10 second (one every 20') lasting only a few seconds and reaching about 50-100 dB.

In such people catecholamines were determined in a day off and after the noise exposure: the former was indicated "BLANK" and the latter "TEST". The analyses were executed according to DOD-FIA technique and the excretion values are reported in mg/l.

As the "TEST" urinary specimen was collected at the end of exposure, and corresponded to the urine flown in blader during all times of exposure, also urinary specimen of "BLANK" was collected for the same time, in the day before the test, in the same subject resting in a noiseless place.
RESULTS

The following table shows the data obtained:

<table>
<thead>
<tr>
<th>Noise Level (dB)</th>
<th>Area &quot;A&quot; (continuous noise)</th>
<th>Area &quot;B&quot; (intermittent noise)</th>
</tr>
</thead>
<tbody>
<tr>
<td>80</td>
<td>120</td>
<td>80-100</td>
</tr>
<tr>
<td>Exposure Duration (h)</td>
<td>1 1/2</td>
<td>9</td>
</tr>
<tr>
<td>Blank (mcg/hr)</td>
<td>4.82 ± 2.91</td>
<td>4.07 ± 4.74</td>
</tr>
<tr>
<td>Test (mcg/hr)</td>
<td>3.36 ± 3.03</td>
<td>4.78 ± 3.00</td>
</tr>
</tbody>
</table>

(We recall that normal catecholamine excretion in other previous research determined, is 3-5 mcg/hr)

At the end of the exposure all the subjects didn’t feel tired and no fatigue symptoms appeared.

CONCLUSIONS

This research has shown that no change happens on catecholamine excretion after a noise exposure for a few hours; all this seems to mean that noise can be heard without any adrenal gland response (perhaps human tolerance can be due to ear protection and noise training).

The catecholamine release was similar in all the workers but one, high duty responsibilities charged, whose "TEST" value was 4 times higher than "BLANK", confirming what we have already achieved in previous experiences, that psychic load can cause catecholamines increase.

DISCUSSION

Q. I believe your method was concerned with the total measurement of catecholamine excretion. Have you obtained no effects by measuring total catecholamine excretion although the results were very variable? Is it possible that if you had measured differential catecholamine excretion such as the separate components of it or that you would have found some changes? Also, some cortico-steroids, at least as far as animal experiments are concerned, are very sensitive to noise. Have there been experiments done in men or is it more appropriate to use cortico-steroids in these types of estimations?

A. Catecholamine excretion is different from the cortico or the adren cortico-steroid reaction because the cortico excretion is quickly responsive to stress while the corticosteroids produce a progressive reaction. Our experiments lasted for a short time and this time no stress reaction was observed. We think that such stress might occur in humans to prolonged exposures at approximately 100 db.
<table>
<thead>
<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>J. F. Pritchett, R. S. Caldwell, R. K. Chesser, J. L. Sartin</td>
<td>Dept. of Zoology-Entomology, Agricultural Experiment Station, Auburn University, Auburn, Alabama</td>
</tr>
</tbody>
</table>

**Citation:**


<table>
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<tr>
<th># of Ref.'s</th>
<th># of Fig.'s</th>
<th>Language</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>2</td>
<td>English</td>
</tr>
</tbody>
</table>

**Type & duration of experiment:**

Study of field exposure to noise; short term mice were collected over a 2-day period and adrenals assayed in 1-day.

**Purpose for study:**

To examine *in vitro* adrenocortical response of wild mice exposed to noise vs. a control group when given ACTH; to study difference in response to noise in domestic and wild mice.

**Description of test groups (subjects, N, age, etc.):**

| 2 groups of wild field mice: Group A - 14 adult male mice collected near the end of a runway at Memphis International Airport; Group B - 9 adult male mice from a control field 2 kilometers from the airport field. |

**Control of other stressors:**

Animals exposed in the field - no control background noise levels for test and controls about 45 dB.

**Noise Stimulus:**

Source: jet aircraft noise.

**Spectral characteristics:** Not specified.

**Noise level:** Average of 110 dB.

**Length of exposure:** Not known but through whole life of each animal.

**# of trials:** Not applicable.

**Statistical Methods:**

Student's t-test; standard error; 2x2 factorial design for analysis of variance.

**Nonauditory effects:**

Adrenal glands assayed *in vitro* for corticosterone secretion rates with and without added ACTH. Basal secretion rates without ACTH were higher in noise-exposed mice than in controls; adrenal response to added ACTH was significantly greater in control than in noise exposed mice.

**CVS Response Measured:**

None.

**Author's conclusions:**

The jet aircraft noise to which the mice in this study were exposed interacted in the body such that the *in vivo* cortical responsiveness to added ACTH was decreased. Previous researchers have found that ACTH did increase the cortical response of adrenals in noise exposed animals. The present results may disagree due to the lack of a pre-incubation period for the adrenals.

**Evaluation & comments:**

More standard and more accurate adrenal assay methods need to be devised so that different studies can be compared more easily.
EFFECT OF JET AIRCRAFT NOISE UPON IN VITRO ADRENOCORTICAL
RESPONSE TO ACTH IN FERAL PIG MUSCULUS

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Ronald K. Chesser1 and James L. Sartin2

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Agricultural Experiment Station
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Auburn, Alabama

(Received in final form December 29, 1979)

Paired adrenals from feral male Mus musculus trapped from fields
designated to the Memphis (TN) International Airport and from suitable
control areas were incubated in the presence or absence of ACTH.
Control animals exhibited lower basal secretion rates of corticosterone
when compared to their noise exposed counterparts. However, ACTH
affected a significantly greater increase in corticosterone secretion
in controls as compared to the noise exposed group. The data suggest
a noise related decline in adrenal cortical responsiveness to ACTH.

A review of the effects of high intensity-noise upon domestic and feral animals
suggests that little is known of specific noise-induced changes in adrenal cor-
tical physiology in feral populations (1). Such changes in domestic and labor-
atory species are better documented.

Otsinaa and co-workers reported a decline in adrenal weight in rats exposed
to 80 dB SPL (sound pressure level) for periods up to 166 days (2). Others
have reported increases in adrenal weight in rats exposed to various types of
noises ranging from 92 to 100 dB SPL for various lengths of time (3, 4). We
have observed significant increases in adrenal weight in feral Mus musculus
exposed to jet aircraft noise both in the field and with laboratory simulation
(unpublished data).

Decreases in numbers of circulating eosinophils have been reported in mice,
rats, and guinea pigs subjected to 74-140 dB SPL for intermittent periods up.
In 12 weeks (2-3), in addition, the depletion of adrenal ascorbic acid has been noted in rats incident to chronic exposure to intense noise (2). Jurtshuk and coworkers have reported an increase in adrenal ascorbic acid with a shorter noise (120 db SPL) exposure time (1-15 days) (2).

Hiroshige et al. have presented data from laboratory rats which indicate a noise-related increase in hypothalamic release of corticotrophin releasing factor (CRF), a substance which in turn elicits the release of ACTH from the adenohypophysis (9). In related studies we have observed both an elevated secretion rate of corticosterone and lack of adrenocortical response to exogenous ACTH in feral Sinonorus hispidus subjected to intermittent jet aircraft noise (100 db SPL) for 4 weeks.

With few exceptions, most reports involving adrenal cortical-high intensity noise interactions have centered upon indirect indices of cortical activity, i.e., adrenal weight, vascular formed element changes, plasma corticoid levels, etc. In many physiological states, such parameters may not accurately reflect the true activity of the gland. Direct determination of secretory activity utilizing in vitro incubation methods may provide a productive alternative approach.

This investigation was designed to detect possible changes in both basal and ACTH stimulated in vitro corticosterone secretion rates of feral animals living in close proximity to a high intensity noise source, i.e., a metropolitan jetport. Since most previous studies have dealt with laboratory species, the present investigation is especially pertinent since Seabloom and Seabloom have recently reported significant differences in both basal and ACTH stimulated corticosterone secretion rates in feral H. musculus as compared to their laboratory counterparts (10). It has been suggested that such differences may be due to a genetically related decrease in adrenal cortical sensitivity in laboratory species, thus raising the possibility of a differential adrenocortical response to noise in comparisons of laboratory with feral species.

Methods

Experimental Animals and Collection Sites

Two groups of animals were utilized in this study. All animals were collected within a 2-day period in February, 1975 and, prior to sacrifice, were housed for 1 day at 26°C, were subjected to a 14-hr photoperiod (0700-2100), and were given Purina lab chow and tap water ad lib. Fourteen adult male H. musculus were collected from a field approximately 90 meters from the end of a runway at Memphis International Airport, Shelby County, TN. Nine adult males of the same species were collected from a control field approximately 2 kilometers from the airport field.

Both fields were similar in habitat in that they were abandoned fields in early stages of secondary succession. Vegetation consisted mainly of grasses and herbs with a few scattered small trees. Additionally, mark-recovery analysis indicated similar population densities at both sites. The close proximity of the two fields insured similarity in genetic composition.

Differential sound levels of both sites were measured with a sound survey meter. Background levels were essentially equal with maximal recorded levels infrequently ranging to 85 db SPL. Noise levels due to incoming and outgoing aircraft at the airport field averaged 110 db SPL. Aircraft noises at the control field were negligible over background.

In Vitro Incubations

All animals were weighed between 9000 and 11500 g. Noise-exposed animals a dish of cold (>4°C) trimmed to remove analytical balance t quartered and incubated 200 ml of liver per 4, and carried out in a Dub oxygen atmosphere. An acid was removed to glass via

Twenty-four hr. late determination of core model 110 fluorometers.

Weight Relationships

Control mice were sacrificed (Table 1). Adrenal pair weights nutrient availability exposed animals since

<table>
<thead>
<tr>
<th>Method</th>
<th>N</th>
<th>Control</th>
<th>Noise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td></td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>Std Error</td>
<td></td>
<td>0.003</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Adrenal Secretory Rate

Secretory data were p and noise, no ACTH an noise and ACTH as well between appropriate m analyzed (Table 1). Gland pair/50 ml, n is unit of glandular weight summarized in Table 2.

Analysis of variance c effects (p < 0.001), an ASR means revealed a n range in noise expo in secretion rates over (26% and 59%, respect
In vitro Incubation and Fluorimetric Determination of Corticosterone

All animals were weighed to the nearest 0.1 g and then killed by decapitation between 1100 and 1110 hours on a single day. Sacrifice order of control and noise-exposed animals was randomized. Adrenal glands were rapidly removed to a dish of cold (-4°C) Krebs Ringer bicarbonate (KRB). The gland pairs were trimmed to remove adipose tissue, blotted on filter paper, and weighed on an analytical balance to the nearest 0.1 mg. Glands from each animal were then quartered and incubated in either 2 ml KRB or a like volume of KRB containing 200 milliunits per ml ACTH (Sigma Biochemicals). Incubations (90 min.) were carried out in a Dubnoff metabolic shaker (10°C, 60 oscillations/min., 5% O₂, 5% CO₂ atmosphere). At the end of the incubation period, incubation media were removed to glass vials and frozen (-20°C).

Twenty-four hr. later, samples were thawed and extracted for the fluorimetric determination of corticosterone (11). Fluorescence was determined with a Turner model 110 fluorometer utilizing #110-B13 (primary) and #110-B18 (secondary) filters.

Results and Discussion

Weight Relationships

Control mice were significantly heavier (p<0.05) than their noise exposed counterparts (Table 1). No significant differences were noted in comparisons of adrenal pair weights or adrenal-body weight ratios. It is unlikely that lack of nutrient availability was related to the smaller body weights observed in noise exposed animals since the habitats were remarkably similar in that respect.

<p>| Table 1 |
| Body and Adrenal Weight Relationships |
|---|---|---|---|
| Control | Noise | |</p>
<table>
<thead>
<tr>
<th>Body Weight</th>
<th>Paired Adrenal Weight</th>
<th>Adrenal Wt./Body Wt.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>9</td>
<td>15.34 ± 0.6 g</td>
</tr>
<tr>
<td>Noise</td>
<td>10</td>
<td>4.10 ± 0.36</td>
</tr>
</tbody>
</table>

Adrenal Secretory Rates

Secretory data were prepared utilizing a two by two factorial design (no noise and no ACTH and ACTH) for the analysis of variance. The main effect of noise and ACTH as well as noise-ACTH interactions were analyzed. Differences between appropriate mean pairs were analyzed with Student's 't' test. Variables analyzed included absolute secretion rate (ASR or units of hormone produced/gland pair/90 min.), relative secretion rate (RRR or units of hormone produced/unit of glandular weight/90 min) and RRR/unit of body weight. The data are summarized in Table 2.

Analysis of variance of ASR revealed significant noise effects (p<0.002), ACTH effects (p<0.001), and noise-ACTH interaction (p<0.001). Comparison of the ASR means revealed a much greater (but insignificant, p>0.15) basal secretion rate in the noise exposed group. ACTH induced a significant (p<0.001) increase in secretion rate over basal levels in both control and noise exposed groups (260% and 59%, respectively). However, the response to ACTH in the noise ex-
TABLE 2

<table>
<thead>
<tr>
<th></th>
<th>Absolute Secretion Rate</th>
<th>Relative Secretion Rate</th>
<th>Relative Secretion Rate/Body Wt.*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
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<tr>
<td>Basal</td>
<td>5.77 ± 1.12 μg</td>
<td>7.73 ± 3.01 μg</td>
<td>0.51 ± 0.23 μg</td>
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<tr>
<td>ACTH</td>
<td>5.59 ± 0.14</td>
<td>15.47 ± 3.39</td>
<td>2.34 ± 0.25</td>
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<tr>
<td>Noise</td>
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<tr>
<td>Basal</td>
<td>0.77 ± 0.09</td>
<td>11.79 ± 3.56</td>
<td>0.83 ± 0.15</td>
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<tr>
<td>ACTH</td>
<td>1.04 ± 0.11</td>
<td>13.71 ± 2.56</td>
<td>1.36 ± 0.10</td>
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*Mean ± Standard Error

Micrograms/Adrenal Pair/30 min.
Micrograms/100 mg Adrenal/90 min.
Micrograms/100 mg Adrenal/g Body Wt./90 min.

NAD, ACTH, and Corticosterone Secretion Rates

The data presented strongly indicate that jet aircraft noise of the type and intensity studied interacts with the organism so as to diminish in vivo corticomedullary responsiveness to exogenous ACTH. Although decreased responsiveness of mammalian adrenal cortices to ACTH may reflect lower endogenous ACTH titers (12), we do not believe the data indicate subcontrol ACTH levels in noise exposed animals. No apparent differences in adrenal weights or adrenal-body weight ratios were observed between control and noise exposed animals. Indeed, the latter group showed elevated basal secretion rates. De Wied, et al. have demonstrated that the in vitro production of corticosterone utilizing a similar incubation protocol (no preincubation period and in the absence of exogenous ACTH) is a reliable index of in vivo cortical activity (13). Since the amount of ACTH bound to the gland at death is responsible for the rate of activity of the gland in vitro, the data suggest slightly elevated ACTH levels may have been present in noise exposed animals.

In vivo responsiveness to exogenous ACTH has been shown to reflect the recent history of exposure of the adrenal cortex to circulating ACTH. Glands from rats subjected to chronic ACTH treatment in vivo produced significantly greater amounts of corticosterone in response to in vitro ACTH stimulation than did glands from animals not subjected to prolonged ACTH administration (14). In addition Bakker and de Wied, utilizing essentially the same incubation methods as employed in our investigation, demonstrated that glands from stressed animals produced substantially more corticosterone in vitro, both in the basal state and when stimulated with given levels of ACTH, than did control glands (15). It was noted, however, that since the basal values of the stressed animals were greater, the percentage increase in steroid production due to ACTH stimulation was not our observations on what noise-exposed rats responded to ACTH than did the type and duration of ACTH responsiveness since there is no influence group, the data tend to group a diminish to ACTH stimulation.

At this point we must clarify the role of ACTH as a potent modulator of responsiveness to corticosterone. It has been shown that such a protocol is a good measure of corticosterone and its levels are taken at the time of the study. However, these levels may be due to the gland at the time of the study. Further studies are necessary to determine the possible effects of non-exposed animals.

In the present study, since all glands were of responsiveness as yet undetermined, differences observed or may have been the result of the study. It is therefore possible to conclude that the results obtained in the study were due to the gland's death and is responsible for the rate of activity of the gland in vitro. The data suggest slightly elevated ACTH levels may have been present in noise exposed animals.

2. V. P. OSINIEVA, N. K. Palewski and L. M. 1970
stimulation was not as great as in glands from non-stressed animals. Although our observations on basal rates are in agreement with those obtained by the latter authors, the data obtained from ACTH stimulated glands are not glands from noise-exposed animals produced significantly less corticosterone when exposed to ACTH than did glands from control animals. This may be due to differences in the type and duration of stressful stimuli studied. Therefore, assuming that ACTH responsiveness of glands from our control animals is representative, and since there is no indication of greater circulating ACTH titers within the control group, the data tend to support the hypothesis that glands from the noise-stressed group had a diminished capacity to increase corticosterone production in response to ACTH stimulation.

As this point we must emphasize that most conventional in vitro techniques for the assay of ACTH responsiveness utilize a preincubation period (30 minutes or longer with no ACTH) after which the adrenal tissue is placed in fresh, ACTH fortified medium. Cortical responsiveness to ACTH is greatly enhanced following such a protocol (16). It has been suggested that the lack of maximal responsiveness of cortical tissue to ACTH stimulation in the absence of preincubation may be due to either (a) competition between endogenous ACTH bound within the gland at the time of death and exogenous ACTH or (b) the presence of a heat labile substance present within the tissue which has the ability to diminish ACTH activity (15). The latter substance was presumed by Bakker and De Wied to be released from damaged cells during quartering of the adrenals although the possibility of non-cortical origin was not excluded (15).

In the present study, ACTH responsiveness was determined without preincubation. Since all glands were handled in a like fashion, we must conclude that the lack of responsiveness to ACTH of glands from noise-exposed animals was due to some as yet undetermined factor(s) or state not present in the control group. The differences observed may have been related to changes within the glands proper or may have been the result of the continuing influence of some extra-adrenal substance. Further insight into the specific source or nature of those differences is not within the scope of the present investigation.

It is therefore possible that noise interaction at some as yet undetermined point so as to decrease the functional reserve capacity of the gland, i.e., the ability to markedly enhance secretory activity in response to physiological stimuli. Should this be the case, a significant segment of the noise-exposed animals' stress response mechanism may have been impaired. Several reports have indicated that intense noise superimposed upon other stresses could have damaging effects and significantly reduce life span, at least in laboratory species (2-7). Since the data presented by Seabloom and Seabloom (10) indicate the adrenal cortical activation system may be more sensitive to stressful stimuli in feral species, these reports assume added significance. In conclusion, there is a significant differential in vitro adrenal cortical response to exogenous ACTH in feral M. musculus exposed to jet aircraft noise as compared with controls. Although the specific site of noise interaction with the adrenal cortex is open to question, it is suggested that such interaction may restrict the animals' ability to respond to physiologically stressful stimuli.

References


The affects high affinity of neuroblasts cell due to environmental changes. Chronic exposure may cause a shift in control values, which cells show early and at the early stage of choline in a biologically active form. The cholinergic effects of ethanol on as to which aspect of ethanol toxicity by ethanol (1), interact signals or from the confusion may be their physiological availability of culture limitations. Presently, the results of neurons maintained under acute and chronic exposure of the initialfinity uptake of choline.

**Cell cultures.** - Neuroblastoma C127, mouse neuroblastosoma C127.
### SUMMARY FORM FOR STUDES ON THE EFFECTS OF NOISE ON THE CARDIOVASCULAR SYSTEM (CVS)

<table>
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<tr>
<th>Principal Investigator(s)</th>
<th>Institution and address where research was performed</th>
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<tr>
<td>A. J. Vander, L. L. Kay, H. E. Dugan, D. R. House</td>
<td>Dept. of Physiology, University of Michigan, Ann Arbor, Michigan 48109</td>
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<th>Purpose for study</th>
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<td>Laboratory experiment; 1-2 hours</td>
<td>The effects of noise on plasma renin activity in unanesthetized rats.</td>
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<th>Description of test groups (subject age, sex, etc.)</th>
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<tr>
<td>Male Sprague-Dawley albinos 200-250 g. One test group was fed a standard Purina chow. The other test group was fed a sodium-free diet; 1 control group.</td>
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<th>Statistical Methods</th>
<th>CVS Response Measured</th>
<th>Nonauditory effects</th>
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<td>handling was minimized</td>
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<th>Noise Stimulus</th>
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<th>Plasma renin activity increased due to 115 dB broadband noise in rats on the normal diet and due to 90-100 dB noise in rats on a sodium-free diet. The 2000 CPS type of noise failed to increase plasma renin activity even over 115 dB.</th>
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<td>source</td>
<td>sound speakers</td>
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<th>Author's conclusions</th>
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<td>Intensities of noise below the human pain threshold can increase plasma renin activity in rats. Plasma renin substrate levels were not altered by noise. The increase in plasma renin activity may be a factor in the development of hypertensive.</td>
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<td>Plasma renin activity due to noise should be studied in humans. Increased renin activity stimulates salt retention leading to water retention, which can create a hypertensive state.</td>
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Effects of Noise on Plasma Renin Activity in Rats

Category: Endocrinology

Arthur J. Vander, Linda L. Kay, Mary Ellen Dugan, and David R. Mouw

Department of Physiology
University of Michigan

Running Title: Noise and renin
Address all correspondence to:

Arthur Vander
6811 Medical Science II
University of Michigan
Ann Arbor, Michigan 48109
1. Research supported by a grant from the Michigan Heart Association.

2. Octave-band analysis of the broad-band noise at 106 dB overall was as follows (all values expressed as dB-SPL): 61 dB at 0.065 kHz; 87 dB at 0.125 kHz; 89 dB at 0.5 kHz; 96 dB at 1 kHz; 101 dB at 2 kHz and 4 kHz; 97 dB at 8 kHz; 82 dB at 16 kHz.
Noise has been shown to induce a wide variety of non-auditory physiological effects (1-3), including persistent hypertension in laboratory animals (4-8). The present study was designed to determine whether acute noise can stimulate renin secretion in the unanesthetized rat.

Methods

All animals used were male Sprague-Dawley albino rats, 200-250 g. They were fed either standard Purina chow (containing 1% NaCl) or a sodium-free diet (Nutritional Biochemicals Corporation) and were housed individually in metal cages; the housing room was on a 6 a.m. to 6 p.m. light-dark cycle. Experiments were performed in separate workrooms; the rats were conditioned to being moved in their cages from the housing room to the experimental sound-box and remaining in the box (with the sound turned off) for five minutes on each of three days preceding the day of the actual experiment; the cages were then returned to the housing room and the animals were removed from the cages and handled briefly. The same six cages were placed in the box together each day and in the same position on the sound-box floor. The sound-box was a sound-proofed wooden box with speakers built into its top.

On the experimental day, six rats were similarly placed in the box, the door closed, and the sound turned on at the desired frequency and intensity for thirty minutes; immediately thereafter, with the sound still on, the rat cages were removed one at a time, and carried to the adjacent room where the rats were decapitated, blood collection lasting thirty seconds. Control animals were subjected to exactly the same sequence of events except that the sound was not turned on. All experiments with animals on the standard diet were performed from 8:30 - 9:30 a.m.; controls and experimentalists were run on consecutive days (the order was rotated from experiment to experiment). The low-sodium animals were studied from 8:30 - 10:30 a.m., the longer time being made possible by the fact
that a low-sodium diet eliminates most of the circadian rhythm for renin (personal observation).

Plasma renin activity (PRA) and plasma renin substrate (PRS) were determined by a modification (9) of the method of Haber et al. (10), which utilizes the generation of angiotension I during a standardized period of incubation and its measurement with the radioimmunoassay kits supplied by New England Nuclear.

All data are presented as means ± one standard error (SEM). The analysis used to determine statistical significance between a set of noise levels and the control for that set was the multiple comparison procedure of Dunnett (11). This method calculates the Student t-statistic in the usual way but then uses the Dunnett tables for P rather than the usual Student t-tables. This is necessary whenever a single group of animals is used as the control for multiple experimental doses (in this case, the multiple noise levels).

Results

Figures 1-3 summarize the effects of broad-band\(^2\) and 2000 cps sounds on PRA in rats on the normal-sodium diet. Broad-band noise caused a significant increase in PRA only when the intensity was increased to 115dB (Fig. 3); 2000 cps sounds failed to increase PRA at any intensity-level studied.

Figure 4 demonstrates that the animals on a low-sodium diet have a greater PRA-response to sound; a highly significant increase in response to broad-band noise occurred at 100dB (P < .01). The response to 90dB was not statistically significant (P = 0.14).

Plasma renin substrate (PRS) was measured in all experiments and no differences were observed between any groups.

Discussion

These data demonstrate that intensities of noise well
below the human pain-threshold can acutely increase PRA in
unanesthetized rats maintained on either a standard or a
sodium-free diet; plasma renin substrate did not change,
indicating that the PRA increase reflects increased plasma
renin concentration. That sodium deprivation reduced the
threshold for this effect is consistent with the fact that
sodium-deprivation enhances the renin-releasing effects of
various stimuli (12).

Given the ability of noise to increase the activity of
the sympathetic nervous system (13,14), it is logical to
postulate that the increased renin secretion is mediated by
increases in circulating catecholamines or enhanced activity
of the renal sympathetic nerves. This pathway has previously
been shown to be involved in the renin response to other types
of stress in the rat (9).

The finding that noise increases PRA may have implications
for the pathophysiology of hypertension. Experimental
hypertension has been produced in laboratory animals using
either pure auditory stimuli (4-6) or mixed auditory-visual-
vibratory stimuli (7,8); the possible role of renin in these
types of hypertension has not been investigated. Finally, it
has been hypothesized that noise may be a risk-factor for
hypertension in people (2,3,14).

Summary

The effects of noise on plasma renin activity (PRA) were
studied in unanesthetized rats. An intensity of 115dB (broad-
band) was required to increase PRA in animals eating a normal
diet; the threshold was only 90-100dB for sodium-deprived rats.
Stimuli at 2000 cps of up to 115dB were ineffective in
elevating PRA.

Acknowledgment The authors gratefully acknowledge the advice
and assistance of Dr. Joseph E. Hawkins.
FIGURE LEGENDS

Figure 1.
Effect of broad-band noise on plasma renin activity in rats on a normal diet.

Figure 2.
Effects of 2000 cps noise on plasma renin activity in rats on a normal diet.

Figure 3.
Effects of 115dB noise on plasma renin activity in rats on a normal diet.

Figure 4.
Effects of broad-band noise on plasma renin activity in rats on a low-sodium diet.
REFERENCES


